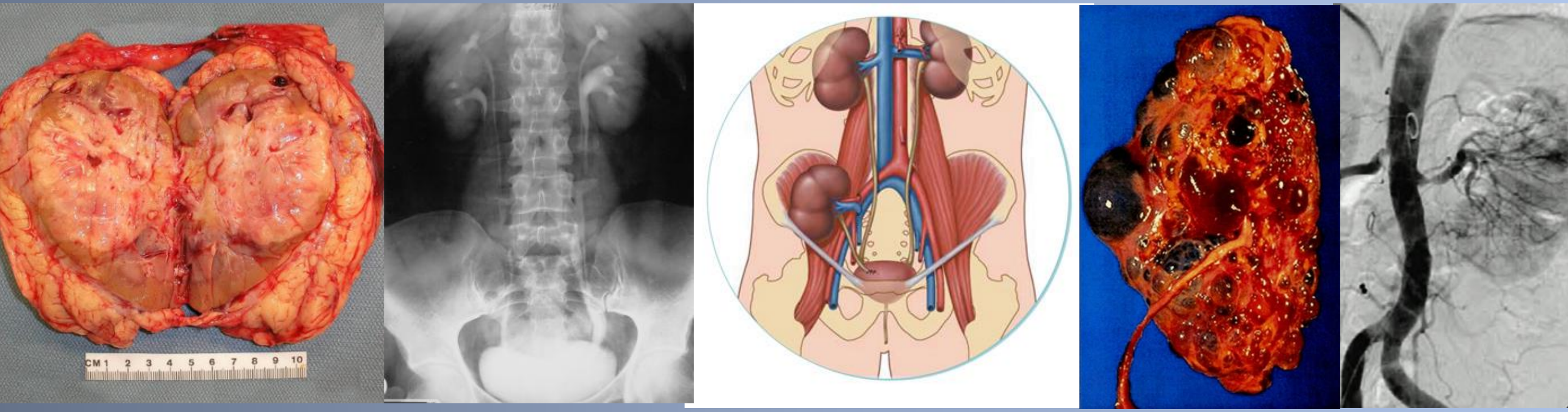
UROLOGY
Picture Questions



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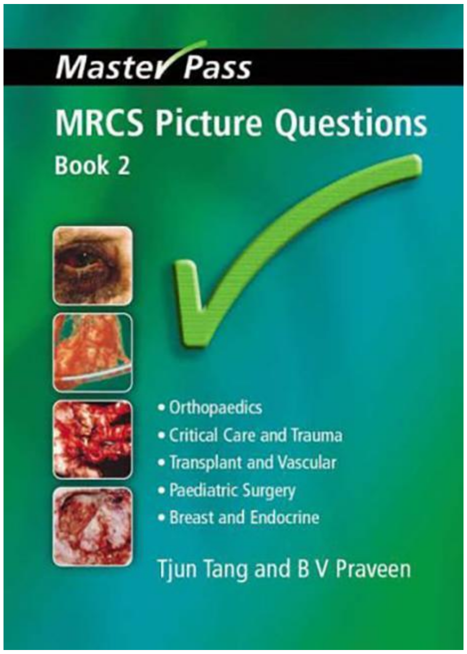


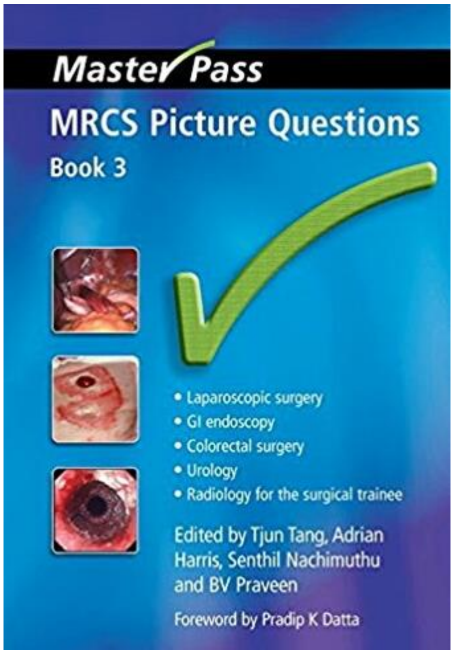
Master Pass

MRCS Picture Questions

Book 2

- Orthopaedics
- Critical Care and Trauma
- Transplant and Vascular
- Paediatric Surgery
- Breast and Endocrine

Tjun Tang and B V Praveen



Master Pass

MRCS Picture Questions

Book 3

- Laparoscopic surgery
- GI endoscopy
- Colorectal surgery
- Urology
- Radiology for the surgical trainee

Edited by Tjun Tang, Adrian Harris, Senthil Nachimuthu and BV Praveen

Foreword by Pradip K Datta

♦ This Book contain Questions in Urology, Kidney Transplantation and few General Questions at the End.

Ahmed Adil

Dr. Ahmed Adil Mohammed

Section 4

Urology

Case 1

A 65-year-old male presented with microscopic haematuria and right loin pain. He underwent a CT scan of his abdomen and pelvis.

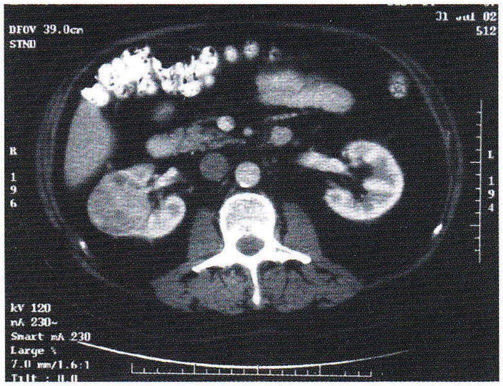


Figure 4.1

Questions

Q1 Describe the findings in Figure 4.1 and the possible diagnosis.

Q2 How would you treat this patient?

Q3 Name some inherited forms of this condition?

Q4 Give some indications for nephron-sparing surgery?

Q5 Name some histological subtypes of this condition?

Answers

(A1) The CT scan shows 5 cm solid tumour arising from the upper pole of the right kidney consistent with a likely diagnosis of renal cell carcinoma. The renal vein, hilar lymph nodes and inferior vena cava are clear of disease. The opposite kidney is normal in terms of size of parenchyma, suggestive that renal function will be normal after nephrectomy.

(A2) Right radical nephrectomy. The procedure can be done through a retroperitoneal (loin) or transabdominal approach. For very large tumours, a transabdominal approach has the advantage of providing adequate space for mobilization to dissect out the hilar vessels. Where expertise is available, the procedure should be done laparoscopically, and if anatomy is favourable (in terms of tumour clearance from the hilar vessels) then a partial nephrectomy is ideal to preserve nephrons. A partial nephrectomy can be done by open or laparoscopical measures, depending on the local expertise of the centre.

(A3) There are four well described types:

- Von Hippel–Lindau (VHL)
- hereditary papillary renal carcinoma (HPRC)
- hereditary leiomyoma renal cell carcinoma (HLRCC)
- Birt–Hogg–Dubé syndrome (BHD). BHD is a hereditary cancer and the affected individuals can have renal tumours, fibrofolliculomas and pulmonary cysts.

(A4) There are some absolute and relative indications for nephron-sparing surgery.

- Absolute indications:
  - tumour in anatomically or functionally solitary kidney
  - bilateral synchronous solid renal tumours.
- Relative indications:
  - selected patients with solid renal mass where contralateral kidney is threatened by local, systemic or genetic conditions that may affect the future renal function, e.g. diabetes, hypertension
  - likelihood of contralateral tumours (hereditary forms of renal cell carcinoma)
  - contralateral normal kidney where tumour size <4 cm (although this size is not absolute)
  - patient preference.

(A5) The various histological subtypes of primary renal cell cancers are:

- classical clear cell (arising from the proximal convoluted tubule)
- papillary (arising from the distal convoluted tubule)
- chromophobe (arising from the cortical portion of the collecting duct)
- collecting duct (very rare, usually in younger patients and carries a poor progosis)
- neuroendocrine
- medullary (arising from the calyceal epithelium, occurs in young, black sickle-cell sufferers and also carries a poor prognosis)
- unclassified.

Case 2

A 45-year-old female had an ultrasound for left loin discomfort. A left renal tumour was found. She subsequently had a CT scan.

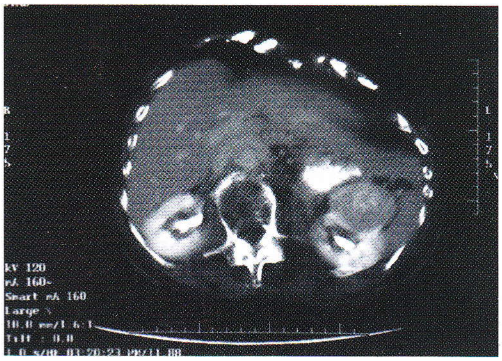


Figure 4.2

Questions

- Q1 Describe the findings in Figure 4.2 and the possible differential diagnosis.
- Q2 What are the radiological appearances of these conditions?
- Q3 How are these conditions treated?

Answers

(A1) There is a 5 cm mass in the anterior aspect of the left kidney suggestive of a renal cell carcinoma (RCC). The possible differentials could be benign renal tumours like an oncocytoma or angiomyolipoma.

Oncocytoma is a benign renal tumour with rare malignant potential. It comprises 3–7% of renal tumours. Men are affected twice as commonly as women. It can occur concurrently with RCC or can mimic RCC radiologically. They may exhibit a classical spoke-wheel sign on CT due to the central stellate scar, but this may also occur in RCC.

Angiomyolipoma is a rare benign tumour of the kidney accounting for 0.3% of all renal neoplasms. It is seen in two distinct groups. It is found in 45–80% of patients with tuberous sclerosis, where tumours are typically bilateral and asymptomatic. Sporadic cases are commonly unilateral and tend to be larger than those associated with tuberous sclerosis.

(A2) The radiographic appearance is determined by the fat content. With ultrasound, tumours appear as well-circumscribed, echogenic and hyperechoic masses. The fat on CT scan appears black and measures –10 to –30 Houndsfield units. Negative Hounsfield units suggest the diagnosis of an angiomyolipoma.

(A3) Angiomyolipoma.
The treatment depends on the size and the symptoms. Nearly 80% of tumours <4 cm are asymptomatic. These can be followed with yearly imaging. Lesions larger than 4 cm with moderate or severe symptoms like bleeding or pain should undergo renal-sparing surgery or arterial embolisation.

Oncocytoma – total nephrectomy as pre-operatively they can be impossible to distinguish from RCC, which may also co-exist.

Case 3

A 45-year-old female developed persistent right loin pain. The CT scan is shown in Figure 4.3.

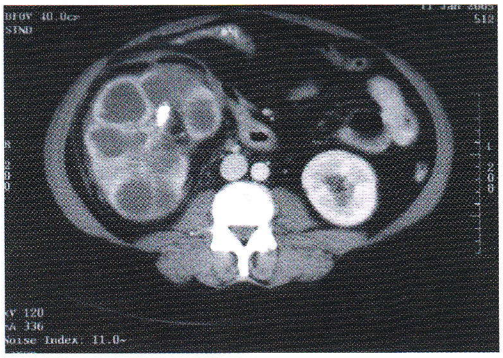


Figure 4.3

Questions

(Q1) Describe the findings on the CT scan.

(Q2) Explain the underlying pathology of this condition.

(Q3) What is the clinical presentation of this condition?

(Q4) How would you manage this patient?

(Q5) What is emphysematous pyelonephritis?

Answers

(A1) The CT scan shows a right kidney with a staghorn calculus in the renal pelvis. There is a thin rim of enhancing parenchyma. The renal fascia is inflamed and thickened with perinephric stranding. The features are those of xanthogranulomatous pyelonephritis.

(A2) Xanthogranulomatous pyelonephritis is as a result of chronic bacterial infection of the kidney. Mostly occurs unilaterally. Severe inflammation and necrosis obliterates the renal parenchyma. Histology is characterised by the presence of lipid-laden histiocytes (xanthoma cells).

(A3) Patients commonly present with flank pain, fever, rigors and persistent bacteriuria. On physical examination, a flank mass can be palpable. *E. coli* or *Proteus* species are commonly cultured from the urine.

(A4) Right nephrectomy, ideally open as it is quite inflamed with obliteration of normal planes due the extensive inflammatory changes. Nephrectomy can be technically more difficult with higher complication rates. During the follow up, patients with recurrent stones may need to have metabolic evaluation to identify the risk of future renal stone formation, although staghorn stones are secondary to recurrent infection by urea splitting organisms and as such do not necessitate a full metabolic stone evaluation.

(A5) It is a necrotising infection of the kidney characterised by the presence of gas within the renal parenchyma or perinephric tissue. Risk factors include diabetes, where a high glucose content can allow the usual organisms (*E. Coli*, *Proteus* or *Klebsiella*) to ferment the glucose to produce carbon dioxide. Mortality in this condition is high and needs urgent treatment.

Case 4

A 70-year-old male presented with frank haematuria. The renal function and haematology results were within normal limits. An intravenous urogram (IVU) was requested followed by an MRI scan.

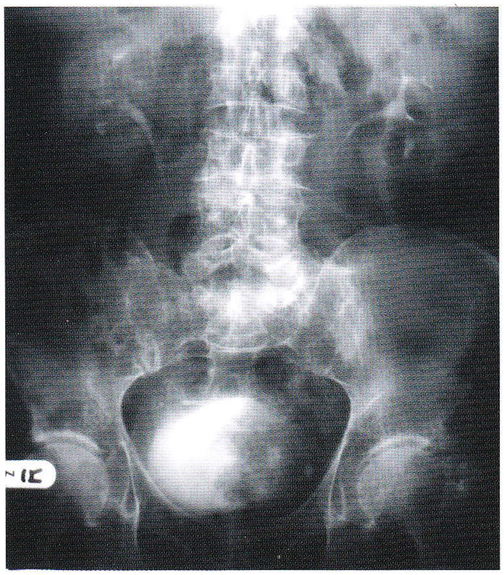


Figure 4.4a

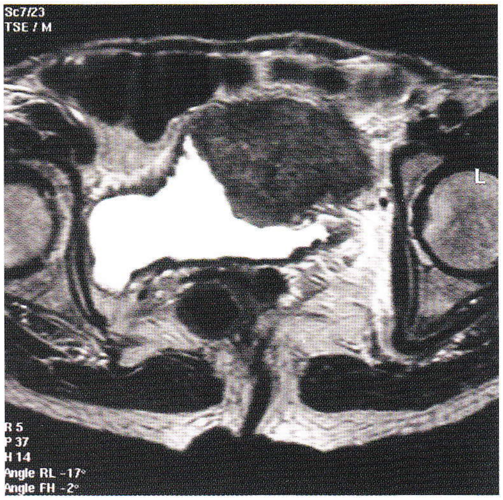


Figure 4.4b

Questions

Q1 Describe the findings and the most likely diagnosis.

Q2 What investigations should be undertaken in a patient presenting with haematuria?

Q3 How do you classify bladder tumours?

Q4 What are the risk factors for developing this disease?

Q5 How do you stage the condition?

Q6 What are the principles of management?

Q7 How are these patients followed up?

Answers

Ⓐ1 The IVU (Figure 4.4a) shows a filling defect on the left side of the bladder. Both kidneys are normal with no evidence of obstruction.

The MRI scan (Figure 4.4b) is a T_2-weighted image (water appears white) and reveals a large (approximately 10 cm) homogeneous mass invading the anterolateral wall of the bladder.

The most likely diagnosis is transitional cell carcinoma of bladder (TCC).

The differential diagnosis for filling defects in the bladder is a large blood clot, tumour and fungal ball.

(A2) The most commonly performed investigations are:

- urine microscopy, culture and sensitivity
- urine cytology
- cystoscopy
- renal tract ultrasound scan.

(A3) Bladder tumours can be classified as primary and secondary tumours.

- Primary:
  - transitional cell carcinoma (>90%)
  - squamous cell carcinoma (5–10%)
  - adenocarcinoma (2%)
  - undifferentiated carcinomas
  - mixed tumours.
- Secondary:
  - local invasion.

(A4) The risk factors for developing bladder cancer are:

- cigarette smoking
- occupational exposure to aniline dyes and aromatic amines (e.g. rubber and dye industries)
- Phenacetin abuse
- Cyclophosphamide chemotherapy.

(A5) Ta – confined to mucosa.
T_1 – invasion into lamina propria.
Tis – carcinoma in situ.
T_2a – invading superficial muscle.
T_2b – invading deep muscle.
T_3a – microscopic invasion of perivesical tissue.
T_3b – macroscopic invasion of perivesical tissue.
T_4a – invasion of prostate, uterus, vagina.
T_4b – invasion of pelvic side wall and abdominal wall.

(A6) The therapeutic options available are:

- non-invasive bladder cancer: endoscopic resection +/- intravesical chemo- or immunotherapy

- invasive bladder cancer: radical cystectomy or radical radiotherapy (still subject to debate)
- transurethral resection of the bladder tumour (TURBT). The procedure is done through a cystoscope. Bimanual examination provides useful information regarding the extent of the tumour. Intravesical mitomycin within the first 24 hours is a standard practice to help reduce the number of recurrences for superficial tumours (up to and including T_1 only).

(A7) Depends on stage and grade. Surveillance cystoscopy every three to six months. Upper tract imaging variable and not always necessary.

Your revision notes

Case 5

This specimen was removed via a cystoscope in a 65-year-old male.

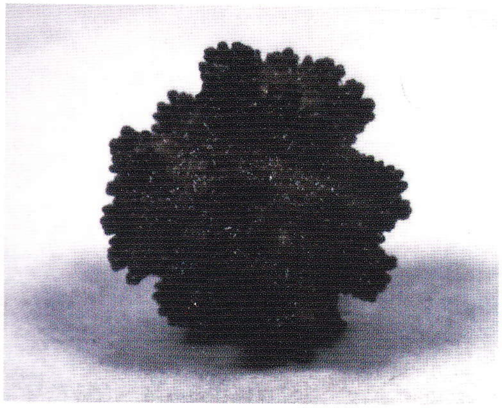


Figure 4.5

Questions

Q1 Identify the specimen and explain why it is this colour.

Q2 How are bladder stones classified?

Q3 What factors predispose to the formation of bladder calculi?

Q4 What is the classical triad of symptoms produced by a bladder stone?

Q5 What investigations would confirm the diagnosis of a bladder stone?

Answers

(A1) The specimen shown is a sharp, spiky bladder calculus.
A calculus is a mass of precipitated solid material in a duct or an organ. The spiky appearance of this calculus is typical of calcium oxalate composition. Oxalate stones are normally white. However this stone is black due to blood pigment deposited on its rough surface as it traumatised the uroepithelium.

(A2) Bladder calculi can be classified as primary and secondary.

- Primary – made up of urate, oxalate, cystine or xanthine owing to an excess of these metabolites in the urine as a result of metabolic abnormalities.
- Secondary – made up of calcium and magnesium ammonium phosphate (triple phosphate) stone.

(A3) Factors predisposing the formation of bladder calculi are:

- anatomical abnormalities – cause stasis in the system or reflux. Examples include a duplex system, urethral valves in newborns and a horseshoe kidney
- stasis – pelviureteric junction obstruction
- infection – especially by *Proteus* species which are urea-splitting organisms that cause an alkaline urine resulting in precipitation and calculus formation
- increased concentration of calcium ions, e.g. in hyperparathyroidism – causes both nephrocalcinosis and calculus formation.

(A4) Urinary frequency, dysuria and haematuria.

(A5) Investigations:

- plain X-ray of KUB (kidneys, ureters, bladder): will invariably demonstrate the stone because of its high calcium content
- cystoscopy: will aid visualisation
- CT urogram: most sensitive investigation for the diagnosis of urinary tract calculi.

Case 6

A 40-year-old male presented to the urological outpatient clinic with this painless scrotal swelling.

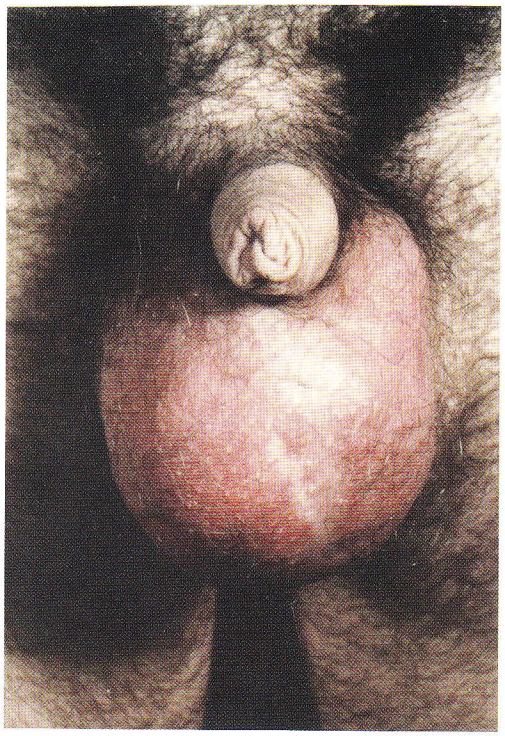


Figure 4.6

Questions

- Q1 What is the most likely diagnosis?
- Q2 What clinical findings would make you suspect this diagnosis?
- Q3 Give a definition of this condition.
- Q4 How is it classified?
- Q5 Is ultrasound useful in the routine assessment of this problem?
- Q6 What are the treatment options?
- Q7 What is the main complication following surgical repair?

Answers

Ⓐ1 Hydrocoele.

A2 Hydrocoele is usually a slowly and uniformly enlarging painless swelling confined to the scrotum with the underlying testis often not palpable through the tense swelling. The swelling is normally firm (tense or lax), non-tender and transilluminates. One can easily get above the swelling.

A3 A hydrocoele is an abnormal collection of serous fluid within the tunica vaginalis of the testis, which forms the outer covering of the testis.

A4 Hydrocoele can be classified on the basis of aetiology and types.
Aetiology: primary and secondary.

- Primary: idiopathic. Appears gradually and becomes large and tense – in children and the elderly.
- Secondary: associated with underlying testicular pathology. Appears rapidly, together with other symptoms.
- Underlying pathologies include:
  - trauma
  - tumour
  - torsion
  - epididymo-orchitis
  - following inguinal hernia repair
  - lymphatic obstruction.

Type: depends on which part of the processus vaginalis remains patent.

- Vaginal hydrocoele: most common type. Processus vaginalis is obliterated and the fluid collects only around the testicle.
- Congenital: the processus remains patent into the peritoneal cavity. Seen in infants. Usually resolves within the first 6–12 months of life but needs surgery if persists after a year.
- Infantile: rare. Processus obliterated at or near the deep inguinal ring but remains patent in the cord and scrotum. Thus the fluid accumulates around the cord as well as around the testicle.
- Hydrocoele of the cord: rarest. Fluid collection is around the cord only. Thus unlike other hydrocoeles, the testicle can be felt separately.

A5 Diagnosis of a hydrocoele is usually clinical and rarely requires an ultrasound scan. However in suspicious cases and in cases of secondary hydrocoele, ultrasound scan can be performed to exclude testicular tumours and identify other underlying causes.

A6 The treatment options can be classified as:

- non-surgical:
  - watchful waiting: if an underlying malignancy has been excluded clinically and with USS, it is reasonable to reassure patient and not operate for asymptomatic, small hydrocoeles.

- surgical:
  - Jaboulay's procedure: the sac is everted and approximated behind the cord via a longitudinal incision
  - Lord's plication: the sac is plicated with a series of interrupted stitches to the junction of the testis and epididymis.

(A7) The main complication following surgical repair for hydrocoele is haematoma formation. Therefore meticulous haemostasis is essential during the procedure. Later it may recur especially if it was large, in which case a Lord's plication may be warranted over a Jaboulay's procedure.

Your revision notes

Case 7

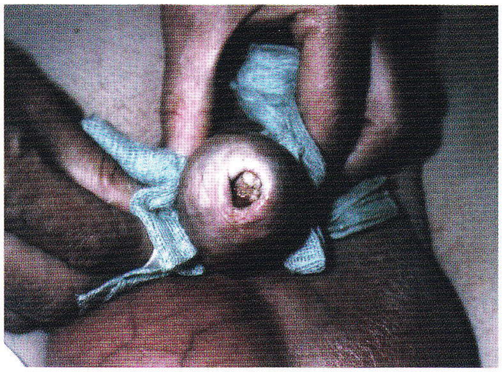


Figure 4.7a

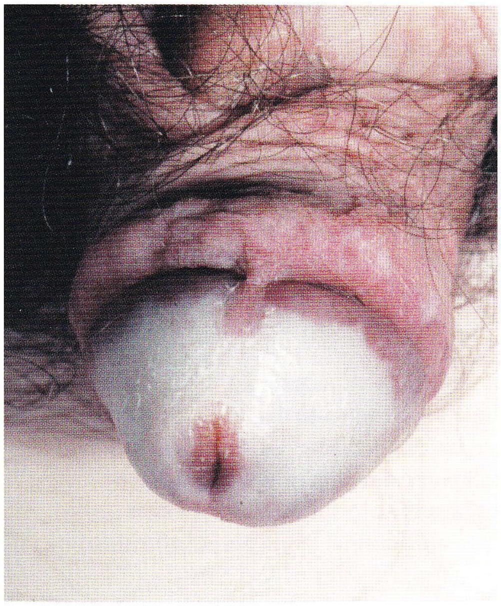


Figure 4.7b

Questions

Ⓠ1 Identify the clinical condition shown in Figure 4.7a.

Ⓠ2 What is the aetiology of this condition? What is the diagnosis in Figure 4.7b?

Ⓠ3 What is the natural history of the prepuce and its function in children?

Ⓠ4 What are the complications of this condition?

Ⓠ5 What is the surgical management and its complications?

Ⓠ6 What is meant by a paraphimosis and how will you manage it?

Answers

(A1) Phimosis: gross narrowing of the preputial orifice. Due to the white scarring of the foreskin, it can be said to be secondary to Balanitis Xerotica Obliterans (BXO).

(A2) The various causes for the development of phimosis are:

- children: congenital/physiological phimosis and scarring due to recurrent balanoprosthitis. BXO may also be a cause
- adult men: balanitis xerotica obliterans (Figure 4.7b).
- elderly: chronic balanitis, most likely associated with diabetes.

(A3) At birth the prepuce is normally adherent to the glans and therefore non-retractile. By puberty it is fully retracted in most boys.

The prepuce is thought to protect the glans and urethral orifice from the excoriation of ammoniacal dermatitis.

(A4) Infection can lead to scarring and fibrosis of the prepuce with consequent narrowing of the orifice. However, in most cases of BXO no preceding history of recurrent infections or inflammation is obtained.

(A5) Management:

- it is important, particularly in children to distinguish physiological from pathological phimosis. Physiological phimosis is to be managed with reassurance and education to the parents, although in some cases topical steroids may be used
- in children, sometimes just the preputial adhesions need to be divided
- true pathological phimosis is best treated with circumcision
- risks/complications of circumcision include infection, bleeding/haematoma, excision of too much skin, injury to the glans penis, breakdown of the wound and in meatal stenosis from extension of BXO scarring to involve the meatus and distal urethra.

(A6) Paraphimosis occurs when a tight prepuce is retracted and fails to return. If it is not subsequently pushed back, the constriction leads to oedema of the glans, pain and occasionally necrosis. This condition needs to be avoided after male catheterisation.

Treatment: manual replacement using gentle pressure with or without local anaesthesia using a penile block. If this is not possible, dorsal slit and then later circumcision after inflammation has settled allowing normal tissue planes and anatomy to be restored.

Case 8

This shows the cut section of a testis which was removed from a 22-year-old male.

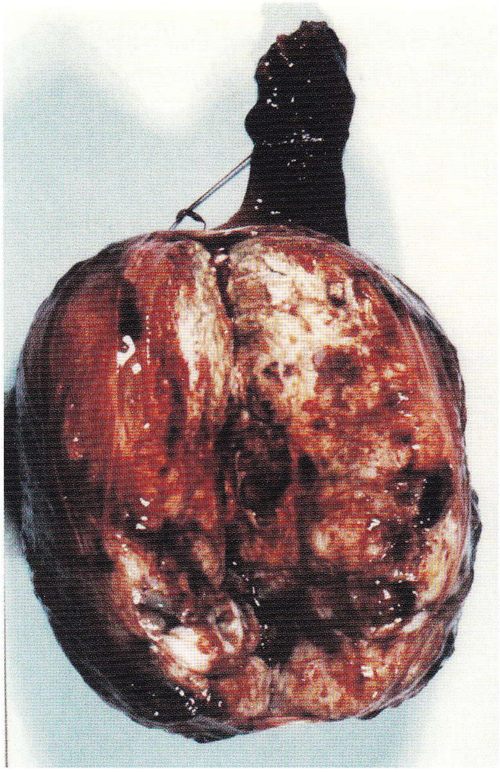


Figure 4.8

Questions

- Q1 What is the most likely diagnosis and why?
- Q2 What are the clinical features of this disease?
- Q3 What is the differential diagnosis?
- Q4 What is the mode of spread?
- Q5 How do you investigate and stage this?
- Q6 What are the treatment options?

Answers

(A1) The diagnosis is malignant teratoma of the testis (Non-semonomatous Germ Cell tumour – NSGCT).

Grossly the specimen shows areas of solid and cystic tissue. Testicular teratoma tends to occur in the younger age group, the peak incidence being 20–30 years. The differential diagnosis is a seminoma of the testis.

Teratoma arises from the totipotent cells in the testis and often contains a variety of cells which may be ecto- or mesodermal in origin. Nodules of cartilage, hair, bone, etc. may be present. Previously was classified on the basis of differentiation as:

- differentiated teratoma
- malignant teratoma intermediate – most common
- undifferentiated or anaplastic teratoma
- malignant teratoma trophoblastic – uncommon.

Seminoma is the commonest testicular tumour and is commoner between the ages of 35 and 45 years. The enlarged testis is smooth and firm. The cut surface is homogeneous and pinkish cream in colour. Histologically there are sheets of oval cells separated by a fine fibrous stroma.

(A2) Symptoms:

- testicular swelling
- sensation of heaviness
- only 10% experience pain
- rarely, may present from metastases to lymph nodes (abdominal mass), lung (haemoptysis, cough), brain (confusion, headaches), bone or liver.

Signs:

- solid scrotal mass which is firm and attached to the testis, but does not transluminate
- may have a secondary hydrocoele.

(A3) The differential diagnosis of testicular tumours includes:

- epididymo-orchitis
- hydrocoele or tense epididymal cyst
- torsion
- syphilitic gumma
- granulomatous/tuberculous orchitis.

(A4) Testicular tumours usually spread via haematogenous or lymphatic route.

- Teratoma spreads by haematogenous route commonly, but also spreads by lymphatics. Pulmonary metastasis may suggest a teratoma.
- Seminoma mainly spreads by lymphatics, whereas haematogenous route is less common. Occasionally, an enlarged supraclavicular lymph node may be the presenting sign.

Lymphatic spread initially involves para-aortic lymph nodes, and later the supra-diaphragmatic or supra-clavicular lymph nodes.

Ⓐ5 Every patient presenting with a testicular swelling must be carefully assessed with the possibility of tumour in mind.

Preliminary ultrasound of the scrotum may be helpful, especially where the testis is not palpable in cases of a large hydrocoele.

Staging investigations should not delay orchidectomy.

Staging investigations include:

- radioimmunoassay for tumour markers – human chorionic gonadotrophin (hCG), alpha feto protein (AFP) and lactate dehydrogenase (LDH) – they help to diagnose, and to assess the effectiveness of treatment
- CT chest, abdomen and pelvis and MRI – to detect secondaries and also to detect response to treatment
- histological analysis of the tumour (high orchidectomy/inguinal approach for orchidectomy with early dissection and soft clamping of the cord prior to delivery of the testis).

hCG levels are raised in 30% of seminomas and 40% of teratomas, but AFP elevation indicates a teratomatous element.

Staging of testicular tumours:
stage 1: testis lesion only – no spread
stage 2: nodes below the diaphragm only
stage 3: nodes above the diaphragm
stage 4: visceral (pulmonary and liver) metastasis.

Ⓐ6 Treatment options:

- teratoma: not radiosensitive:
  - stage 1 can be managed by either chemotherapy to treat the 20% of patients who may have occult micrometastases to para-aortic lymoh nodes or surveillance by regular tumour markers and CT scans on the basis that treating all patients with chemotherapy to target the 20% of these patients that may have micrometastasis is probably over-kill
  - stages 2–4 can be treated with chemotherapy. Ideally BEP – bleomycin, etopside and Cisplatin, although methotrexate, and vincristine have been used especially as second or third line with good results.
- seminoma: radiosensitive:
  - radiotherapy was the mainstay of treatment in stage 1 and 2 disease. More recently trials have shown that for stage 1 disease, 1 dose of carboplatin is as effective as radiotherapy to para-aortic nodes. BEP is still used for stage 2 disease
  - stages 3 and 4 are treated with chemotherapy.

Retroperitoneal lymph node dissection is sometimes needed if the masses remain after chemotherapy especially for NSGCT.

Prognosis depends on the histological type and stage of the disease.

- Teratoma – In stage 1 and 2, the five-year survival is 85%. It drops to 60% in advanced disease.
- Seminoma – If there is no metastasis, 95% will be alive at five years after treatment. It drops to 75% with extensive visceral secondaries.

Your revision notes

Case 9

Figure 4.9 is from a 55-year-old male.

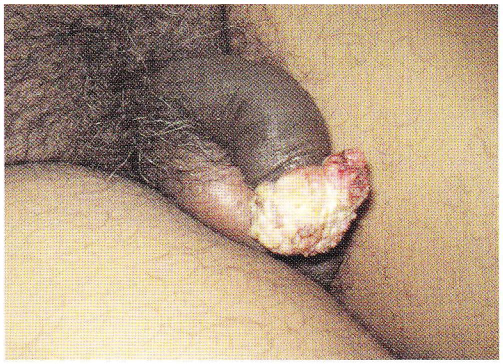


Figure 4.9

Questions

- (Q1) What is the most likely diagnosis?
- (Q2) What are the potential causes and predisposing factors?
- (Q3) What pre-malignant changes are associated with this condition?
- (Q4) What is the clinical course of this disease?
- (Q5) What are the treatment options?

Answers

(A1) Invasive squamous cell carcinoma of the penis.

(A2) The causes and predisposing factors are:

- presence of a foreskin and poor hygiene – accumulation of carcinogens within smegma under the foreskin
- HPV 16 and 18
- smoking
- pre-malignant conditions, e.g. Bowen's disease or erythroplasia of Queyrat, Leukoplakia.

(A3) The premalignant conditions of penile cancer are:

- leukoplakia
- erythroplasia of Queyrat (carcinoma in situ)
- cutaneous horn – overgrowth and cornification of the epithelium.

(A4) Clinical course:

- typically presents as epithelial thickening of the glans or the inner surface of the prepuce progressing to ulcero-infiltrative or exophytic growth eroding the penile tip, shaft or both
- usually grows slowly with metastasis to the regional lymph nodes (inguinal and iliac)
- distant metastasis are uncommon especially if lymph nodes are clear
- five-year survival rate is 66% if lesion confined to penis, but only 27% with regional lymph node involvement.

(A5) The treatment options available are radiotherapy, surgery and chemotherapy.

- Small localised lesions can be treated with external beam radiotherapy, topical treatment with 5FU or imiquimod.
- Large lesions can be treated with surgery:
  - glans resurfacing or glansectomy
  - partial penile amputation if at least 2 cm of residual penis length can be preserved after giving a reasonable margin clearance (used to be 2 cm, but now less is accepted)
  - total penile amputation with perineal urethrostomy can be performed.
- Ilioinguinal lymph node dissection can be performed in case of involved regional lymph nodes or in cases of higher local stage and grade.
- Fixed lymph nodes – palliative treatment with chemotherapy.
- Metastatic disease – chemotherapy.

Case 10

A 65-year-old male was admitted with sudden onset of right loin pain radiating to the groin. On examination, he was febrile and tachycardic. The white count was elevated. The X-ray of his abdomen is shown in Figure 4.10a.

A 55-year-old male was admitted with sudden onset of colicky right loin pain radiating to his groin. His abdomen was not peritonitic and his urine test is positive for microscopic haematuria. Figure 4.10b shows an unenhanced CT scan of his abdomen and pelvis.

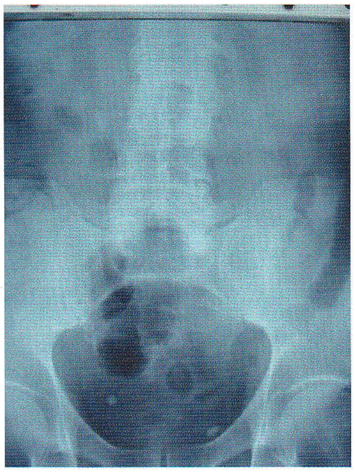


Figure 4.10a

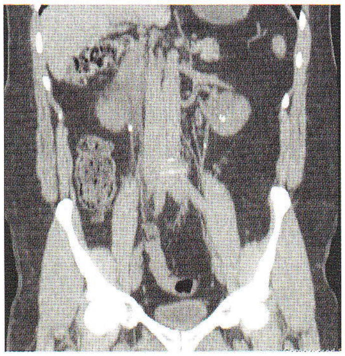


Figure 4.10b

Questions

(Q1) Describe the findings of Figure 4.10a and Figure 4.10b.

(Q2) Apart from an unenhanced CT scan, what investigation would be appropriate after a plain X-ray?

(Q3) What is the most appropriate clinical management of a patient with a dense nephrogram and complete obstruction on an IVU?

(Q4) What are the clinical features of renal colic?

(Q5) How would you confirm your diagnosis?

(Q6) How would you manage renal tract calculus disease?

(Q7) How do you classify renal stones?

(Q8) What is a struvite stone?

Answers

(A1) Plain X-ray (Figure 4.10a) shows a 1.5 cm stone in the right ureter adjacent to the transverse process of L3. Other calcific areas in the pelvis probably represent phleboliths. Phleboliths represents calcifications within the pelvic veins. They are rounder, lateral to the course of the ureter, and have radiolucent centres. A leaking abdominal aneurysm can also present like this. Careful clinical examination is very important. Sometimes a calcified aneurysm wall can be seen on the plain X-ray.

The CT scan image with reconstruction (Figure 4.10b) shows a 6 mm stone in the right upper ureter and a 5 mm stone in the left mid-ureter. Two stones are also seen in the left kidney. The diagnosis is renal colic.

(A2) An intravenous urogram. Some centres use CT scan as the gold standard in patients with renal colic. CT scan is more sensitive and specific than an IVU.

(A3) In an acute setting, ureteric stenting or nephrostomy is carried out as an emergency. Stone removal is carried out as a second stage. Most centres prefer extracorporeal shockwave lithotripsy (ESWL) for stones in the proximal ureter (above the iliac vessels). Distal ureters are managed with ureteroscopes. This man had an emergency stenting.

(A4) Symptoms:

- sudden onset colicky abdominal pain radiating from loin to groin, sometimes to the scrotum, tip of penis or labia, often associated with nausea and vomiting
- difficulty in micturition
- frequency and or urgency in micturition
- septic symptoms, haematuria.

Signs:

- patient cannot lie still due to pain
- urine analysis reveals microscopic haematuria (although the longer the history, the lower the likelihood of finding haematuria and so negative dipstick does not rule out renal colic).

Patients can also present with obstructive uropathy leading to:

- hydronephrosis
- pyonephrosis
- septicaemia
- end-stage renal failure secondary to bilateral renal calculus disease or in a solitary functioning kidney.

(A5) Other investigations:

- urinalysis will reveal microscopic haematuria
- plain abdominal X-ray reveals 90% of renal tract calculi
- CT-Urogram is the investigation of choice as it is extremely sensitive in identifying renal tract calculus. It can also reveal associated obstructive changes, e.g. pelvicaliceal dilatation, hydroureteronephrosis, perinephric stranding
- intravenous urogram and ultrasound can be performed but CT-Urogram is the investigation of choice.

Ⓐ6 Management depends on the following factors:

- size of the calculus
- site of the calculus
- presence of renal tract obstruction/infection
- renal function.

Initial management consists of adequate pain relief, and renal function assessment.

- Calculus less than 6 mm in size will pass on its own, especially if in distal ureter and only needs expectant management with analgesia and alpha receptor antagonists, which relax lower ureteric smooth muscle and has been shown in meta-analysis to increase stone passage rates.
- Calculus larger than 6 mm will need intervention, the timing of which depends on renal function, renal tract obstruction and sepsis.
- If renal function satisfactory and no obstruction or sepsis, patient can be managed conservatively initially, followed by early elective intervention in the form of ureteroscopic extraction or ureteric stenting and ESWL (Extracorporeal Shock Wave Lithotripsy).
- In cases of renal tract obstruction, sepsis, worsening renal function or patients with solitary kidney, emergent intervention is required in the form of percutaneous nephrostomy or ureteric stenting.

Ⓐ7 Renal stones can be classified as:

- calcium oxalate stones (85%)
- struvite stones (infective or triple phosphate stones) in 15%
- uric acid stones (5–10%)
- cystine stones (1–5%) – inherited as autosomal recessive trait.

Ⓐ8 Struvite stones are infectious stones made of calcium magnesium ammonium phosphate. They are more common in women (2:1). Alkaline urine caused by breakdown of urea (by urease produced by *Proteus, Pseudomonas* and *Klebsiella* spp.) paves way for the formation of these stones. A pH >7.0 is needed for stone formation. The stones can fill the entire renal collecting system (staghorn stones).

Your revision notes

Case 11

This 45-year-old male presented with several scrotal masses, which were separate from the testes and felt firm and loculated. Figure 4.11a shows the ultrasound of one of these masses.

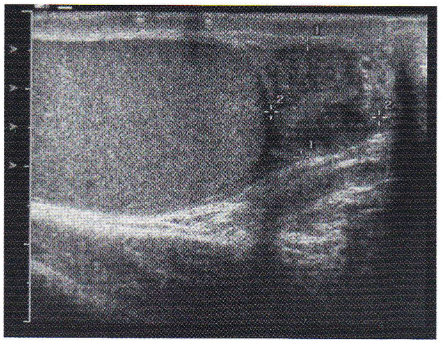


Figure 4.11a

Questions

- Q1 What is the most likely diagnosis?
- Q2 How are they caused?
- Q3 What are the defining features?
- Q4 What are the management options?
- Q5 What risks should you warn the patient about if they undergo removal?
- Q6 Look at Figure 4.11b. What is the spot diagnosis?

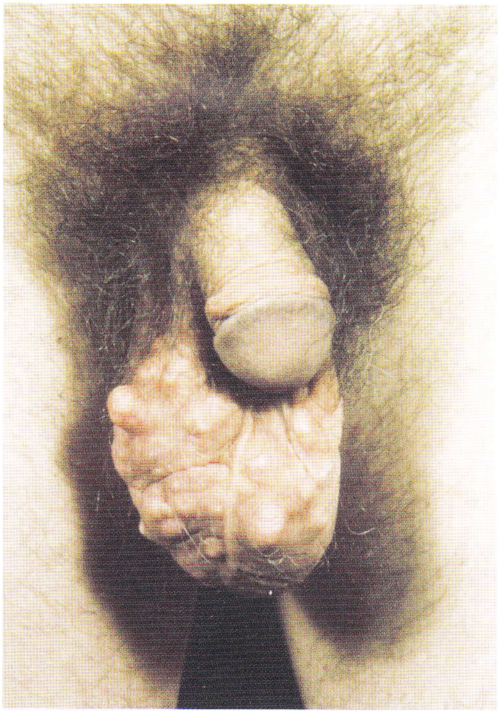


Figure 4.11b

Answers

(A1) Multiple epididymal cysts.

(A2) Occasionally they occur as a complication of vasectomy. In these cases they are full of sperm and are termed spermatocoeles.

(A3) Mass(es) are often multiple and most commonly arise in the head of the epididymis – separate from the testis.
Firm and may be loculated.
Brilliantly transilluminable (classical) unless they contain sperm.
Separate from the superficial inguinal ring (can 'get above' mass(es)).

(A4) Non-surgical versus surgical.

- Non-surgical: if asymptomatic they should be left alone especially in younger males where there is a risk of subfertility if operated upon, since epididymal obstruction can occur. An alternative to surgical removal is aspiration, though recurrence is usual.
- Surgical: very large or painful cysts can be removed. Occasionally the whole of the epididymis is excised to prevent recurrence.

(A5) BAUS procedure specific consent form – recommended discussion of adverse events.
Occasional:

- recurrence of fluid collection can occur
- collection of blood around the testis – can resolve slowly or require surgical removal
- infection of incision or testis requiring further treatment.

Rare:

- scarring can damage the epididymis causing subfertility.

Alternative therapy:

- observation
- removal of fluid with a needle.

(A6) Multiple scrotal sebaceous cysts.

Case 12

A 60-year-old man presented to the GP with lower urinary tract symptoms. The PSA was 19.5. He underwent this investigation (Figure 4.12).

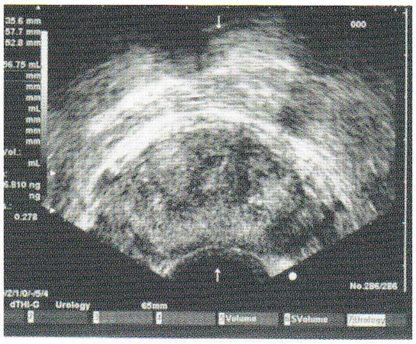


Figure 4.12

Questions

- Q1 What is the investigation? What are the possible complications of this procedure?
- Q2 What does it show? How is prostate cancer graded?
- Q3 Give the treatment options available.
- Q4 What are the indications to perform the prostate biopsy?
- Q5 Name a recent advance in managing this condition.

Answers

(A1) The picture shows a transrectal ultrasound of the prostate gland. Biopsies are taken under transrectal ultrasound guidance. The procedure is usually done under local anaesthesia. Complications of this procedure include haematuria, urinary retention, bleeding per rectum, haemospermia. Mortality secondary to sepsis has also been reported following this procedure.

(A2) TRUS shows a hypoechoic area in the peripheral zone of the prostate likely to represent a malignancy (prostate cancer).

Gleason grading.
It is based on the glandular pattern of the tumour as identified at relatively low magnification. Cytologic features play no role in the grade of the tumour. The primary (or the most predominant) and the secondary (or the second most prevalent) architectural patterns are assigned a grade from 1 to 5, with 1 the most differentiated and 5 the least differentiated. The total gives the Gleason score or the sum.

(A3) For localised disease:

- radical prostatectomy – open (retropubic or perineal) or minimally invasive (laparoscopic or robotic assisted)
- radical radiotherapy
- brachytherapy
- watchful waiting.

For locally advance or metastatic disease:

- hormone deprivation treatment.

(A4) Abnormal digital rectal examination.
PSA elevated more than the age specific range – PSA is a serine protease secreted by the prostatic epithelium.
Note false positive PSA elevation can occur in BPH, prostatitis, recent sexual activity.

(A5) Brachytherapy – interstitial radiotherapy used for managing organ-confined prostate cancer. Suitable for smaller prostate with low volume cancer in men with minimal lower tract symptoms.
Robotic prostatectomy.

Case 13

This 35-year-old male presents with intermittent right loin pain. On examination, he was afebrile with right loin tenderness. Plain X-ray abdomen did not reveal any renal stones. An IVU was requested (Figure 4.13).

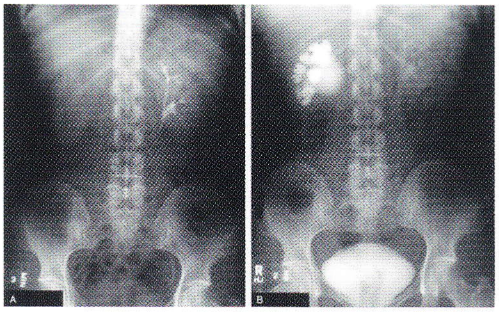


Figure 4.13

Questions

- (Q1) Describe the IVU findings and the diagnosis.
- (Q2) What further investigations are needed?
- (Q3) Name some complications of this condition.
- (Q4) How can this condition be treated?
- (Q5) Give some of the recent advances in managing this condition.

Answers

Ⓐ1 IVU shows dilatation of the right renal pelvis with a normal ureter in keeping with pelviureteric junction obstruction (PUJ). This condition is probably the most common congenital abnormality of the ureter, occurring more often in boys than in girls (5:2 ratio). It is more common on the left side (5:2) and can be bilateral in 10–15% of the cases.

Ⓐ2 Isotope renogram with Technetium-labelled mercapto acetyl guanine (MAG 3) to assess the degree of obstruction. Additional diagnostic tools include spiral CT, transluminal ultrasound, and Whitaker's test.

Ⓐ3 Recurrent UTI, stone formation, sepsis, loss of kidney function.

Ⓐ4 Anderson–Hynes dismembered pyeloplasty. The purpose of this procedure is to remove the narrowed portion of the PUJ and attach the ureter to the renal pelvis in a way that allows easy drainage of urine.

Ⓐ5 Laparoscopic pyeloplasty – non-randomised studies show equal efficacy with this procedure whilst avoiding the loin scar and reducing the hospital stay.

Endoscopic procedures:

- retrograde endopyelotomy
- balloon rupture
- antegrade endopyelotomy
- stent insertion (retrograde or antegrade) with six-monthly to yearly stent changes.

Case 14

A 15-year-old boy presented to A&E with a four-hour history of left-sided testicular pain. There was no history of systemic illness, testicular trauma or urinary symptoms. Examination revealed mild scrotal erythema, a high-riding and tender left testis.

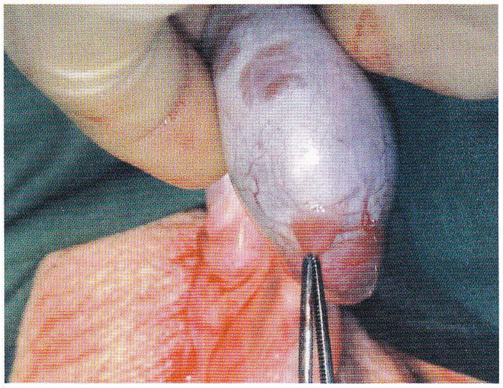


Figure 4.14

Questions

Q1 What is the most likely diagnosis and why? What is the differential diagnosis?

Q2 What investigations may be performed to support this diagnosis?

Q3 Figure 4.14 was taken during exploration of this boy's left testicle. What do you see?

Q4 How would you manage the patient?

Q5 Suppose that this patient in fact had only a torted hydatid cyst of Morgagni, would you explore the contralateral side?

Answers

(A1) Most likely diagnosis is acute left testicular torsion.
Reasons:

- left testicle is higher than the right one ('high-riding'). Usually also lies in a horizontal plane. This is consistent with twisting and shortening of the spermatic cord
- testicular torsion can occur at any age, but most common in boys aged 13–17 (prevalence 1/125 males)
  - an abnormally high investment of the tunica vaginalis allows excess mobility of the testicle (the classical 'bell-clapper' deformity)
  - incomplete attachment of the testes to the epididymis allows it to rotate alone – less common
  - trauma and exercise can produce the cremasteric reflex. This can precede the torsion and some patients recount previous episodes of similar pain due to recurrent torsions that spontaneously resolve
- remember, patients with cryptorchidism are at increased risk of torsion and also young patients with abdominal pain should have their external genitalia examined for this condition.

Differential diagnosis:

- torsion of a hydatid cyst of Morgagni
- acute epididymo-orchitis (rare)
- strangulated inguinal hernia
- idiopathic scrotal oedema.

(A2) None.
Diagnosis is made on clinical grounds. Urgent surgical exploration is required if there is any doubt about the diagnosis!

There is normally a time window of four to six hours from the onset of pain where testicular salvage is possible – after this the testicle has a much higher chance of loss. In some instances even a shorter delay leads to loss of the testicle; therefore speed in exploration is critical!

However:

- urinalysis – to exclude UTI causing epididymo-orchitis
- gray scale ultrasound – may show the affected testicle to be enlarged and hypoechoic. In contrast, in epididymitis the testis is normal
- colour Doppler ultrasonography – used to assess testicular blood flow. Sensitivity and specificity 95% (but operator dependent).

(A3) A torted left testicle (note the discolouration of the testicle).
Forceps is holding an incidental hydatid cyst of Morgagni, which is not torted.

(A4) Points to consider:

- informed consent for bilateral scrotal exploration, possible ipsilateral orchidectomy, and contralateral orchidopexy should be obtained in all cases from parents and if appropriate the patient

- if testicular torsion, untwist spermatic cord and wrap testis in a warm saline soaked swab before deciding on its viability (for 10 minutes). Ask the anaesthetist to increase the ventilated oxygen!
- if viable after assessment, return testis to scrotum and suture to dartos muscle at three different points to prevent recurrence. The other testis is similarly fixated either through the same incision or a separate one
- if orchidectomy required – in order to prevent testicular gangrene or scrotal abscess or prevent the formation of anti-sperm antibodies that would render an adult with a single testis infertile – get a second opinion from your consultant
- if a torted hydatid cyst of Morgagni is found this is excised or diathermied and the testis is fixated. Similarly (testis fixation) if epididymo-orchitis found, except culture swab also taken and patient commenced on antibiotics
- in these two circumstances the testis is fixed to prevent diagnostic confusion in future if the boy represents with testicular pain.

(A5) No – not necessary if pain is unilateral and a torted hydatid cyst of Morgagni is found. What is more difficult is the scenario where the symptoms are suggestive but on exploration, no abnormality whatsoever is noted. This may be due to intermittent torsion and so both sides should be fixed.

Undescended testis

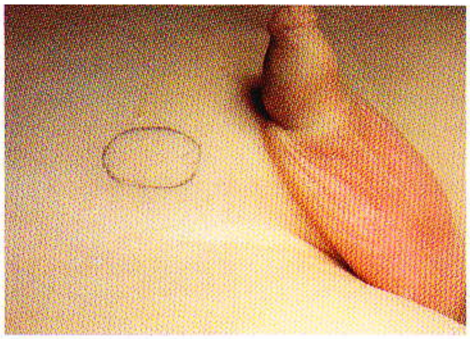


Figure 4.11

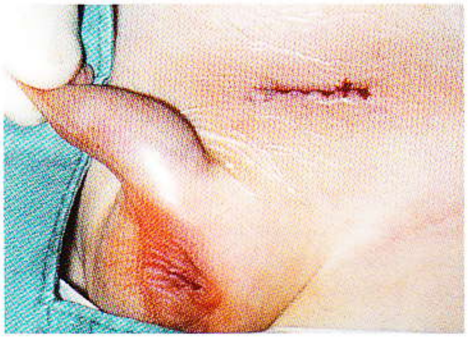


Figure 4.12

A 2-year-old boy is about to undergo surgery following an observation made on his genitalia and a lump in his right groin (Figure 4.11).

Questions

(Q1) What is the most likely diagnosis?

(Q2) What is the differential diagnosis?

(Q3) Can these conditions be distinguished clinically?

(Q4) Where is the right testis lying in this case and how do you know that it is not within the inguinal canal?

(Q5) What are the complications of this condition?

(Q6) Figure 4.12 shows the completed operation on another patient. What has been performed?

(Q7) Are there any useful investigations to perform prior to surgery? Does the fact that the testis is ectopic (not in the normal line of descent) rather than maldescended change your management?

Answers

(A1) Undescended testis/ectopic testis.

(A2) The differential diagnosis is a retractile testis.

(A3) A retractile testis is a normal testicle where an excessively active cremasteric reflex draws the testis up to the superficial inguinal ring. On careful examination in a warm atmosphere it can be coaxed into the scrotum. An ectopic testis cannot be manipulated into the scrotum. If the testis is retractile, the scrotum is normally developed. No further intervention is required. The testicle is likely to descend normally by puberty. Follow-up is mandatory.

(A4) The testis is easily visible and therefore is lying in the superficial inguinal pouch having emerged from the external ring. If it were still lying within the inguinal canal, it would not be certainly visible and probably not palpable.

(A5) Complications of this condition include:

- defective spermatogenesis – sterility if bilateral
- increased risk of torsion
- increased risk of trauma
- inguinal herniation (associated in 90%)
- increased risk of malignant change (relative risk of 20–40%), which appears to be the case even if surgical correction is performed.

(A6)

- Orchidopexy: mobilisation and placement of the testis into the scrotum. This should be carried out as soon as possible. Remember '2 in the bag by the age of 2'! The co-existing inguinal hernia sac should be removed at the same time.
- Exploration is via a high inguinal incision allowing first the inguinal canal then the abdominal cavity to be explored.

(A7)

- Since surgery is always indicated, investigations such as USS (unreliable), MRI and CT (needs anaesthetic) and laparoscopy (normally leads to open surgery) are not indicated.
- Management is as for undescended testis. If a testis is located and found to be dysplastic it must be removed. If normal it must be replaced into the scrotum. There is no indication for testicular biopsy.

Hypospadias

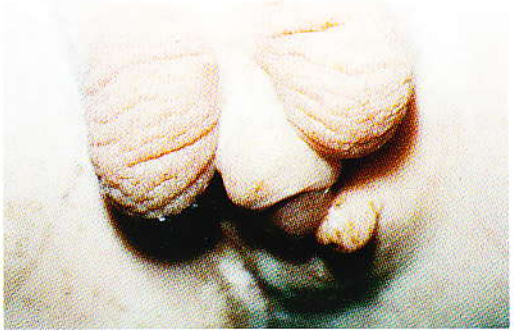


Figure 4.14

Questions

- (Q1) What is the diagnosis?
- (Q2) What has failed during development?
- (Q3) What are the principle components of this condition?
- (Q4) How do you classify this condition?
- (Q5) What is important to establish on presentation?
- (Q6) What is the treatment? What are the potential complications?

Answers

(A1) Hypospadias: a common anomalous development of the distal urethra, penile shaft and glans penis affecting 1 in 400 boys.

(A2) The male urethra is formed by the in-rolling of the genital folds. In hypospadias the urethral plate and groove fail to complete the process of tubularisation and fusion.

(A3)
- The urethra opens onto the ventral surface anywhere from the glans to perineum.
- Ventral curvature (chordee) of the penile shaft due to the presence of fibrous tissue within the penile fascia.
- Deficient ventral foreskin and excess hooded dorsal foreskin.

(A4) It can involve the glans alone or any degree of the penile and perineal urethra. Classification is as follows:
- glanular
- distal/mid/proximal penile
- scrotal
- perineal (15%).

The more proximal position of the urethral orifice makes surgery more difficult and complex.

(A5) On presentation it is important to establish whether hypospadias is an isolated anomaly or whether other urinary tract anomalies co-exist. This is unusual with the more usual glanular/distal penile cases. With more proximal abnormalities, associated anomalies need to be excluded with ultrasound. Other common anomalies in this condition are inguinal hernias and an undescended testis.

(A6) Treatment depends on the site of urethral orifice:
- glanular hypospadias: meatal advancement and glanuloplasty (MAGPI). This can be done as a day case procedure and does not require catheter drainage
- more complex problems may require onlay graft and island flap urethroplasty involving release of the chordee and construction of a neourethra using imported tissue.

Potential complications of surgery are:
- skin ischaemia
- haematoma formation
- infection
- fistula formation
- urethral stenosis
- residual chordee.

Cushing's syndrome

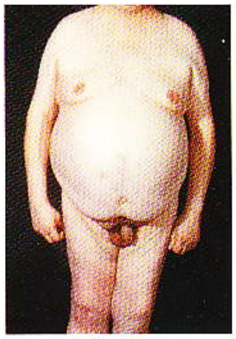


Figure 5.18

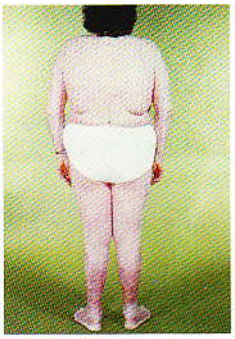


Figure 5.19

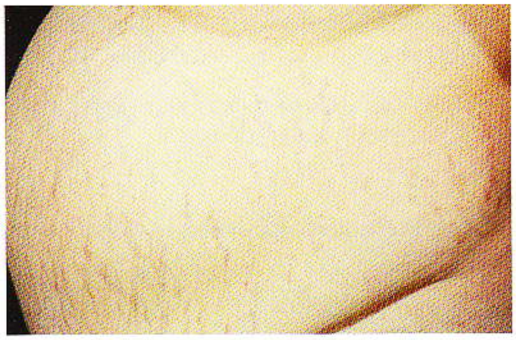


Figure 5.20

A 38-year-old patient presents with a history of recent change in appearance along with weight gain. He also mentioned noticing bruises, which he could not explain. He was found to be hypertensive.

Questions

- (Q1) What is the likely diagnosis?
- (Q2) What is the microscopic anatomy of the adrenal gland?
- (Q3) What are the substances produced by these cells?
- (Q4) What are the causes of Cushing's syndrome?
- (Q5) What are the typical clinical features seen in Cushing's syndrome?
- (Q6) What are the investigations useful in diagnosis and management?
- (Q7) What are the important aspects of management?
- (Q8) What are the surgical problems associated with this condition?

Answers

A1 Cushing's syndrome.

A2 The adrenal gland consists of a cortex (90%) and medulla (10%) which are developmentally and functionally separate. The cortex consists of three layers: an outer zona glomerulosa, middle zona fasciculata and an inner zona reticularis. The medulla consists of chromaffin cells and preganglionic autonomic nerve fibres from renal and coeliac plexus.

A3
- Three classes of corticosteroids are produced by the cortex: mineralocorticoids, glucocorticoids and adrenal androgens.
- The zona glomerulosa produces mineralocorticoids such as aldosterone. Aldosterone acts on the renal distal convoluted tubules to cause sodium reabsorption and excretion of potassium and hydrogen.
- The zona fasciculata and zona reticularis produce glucocorticoids. Cortisol is the main glucocorticoid and has a role in the metabolism of glucose, fat and protein. It increases blood glucose levels.
- The zona fasciculata and zona reticularis also produce androgens. Dehydroepiandrosterone is the main adrenal androgen. It is a weak androgen and the effects only become apparent in adrenal hyperactivity.
- The chromaffin cells of the medulla sythesise, store and secrete catecholamines. Adrenaline, noradrenaline and dopamine are the principal catecholamines. Noradrenaline is the neurotransmitter for the autonomic nervous system. Adrenaline and noradrenaline are present in the adrenals in the ratio of 4:1 and have a half-life of only 1–2 min. Their metabolites metanephrines and VMA are excreted in the urine.

A4 The causes of Cushing's syndrome are divided into two broad groups:
- *ACTH dependent*: pituitary/hypothalamus-dependent Cushing's disease, ectopic ACTH-secreting tumours and therapeutic excess with ACTH or analogue
- *non-ACTH dependent*: benign and malignant adrenal tumours, macronodular cortical hyperplasia, primary pigmented nodular cortical disease, glucocorticoids or analogues.

A5 Weight gain, change in body configuration (lemon on sticks appearance), moon facies, skin pigmentation, thin limbs, wasting of muscles, bruising and striae (Figure 5.20), hypertension, osteoporosis, polyuria, impaired glucose tolerance, amenorrhoea, hirsutism, frontal balding, acne, growth retardation, depression, psychosis, venous thrombosis.

A6 There are three stages of investigations: to confirm hypercortisolism, to determine whether it is ACTH dependent, and imaging to localise the anatomical and pathological abnormality.

Tests to confirm hypercortisolism
- Overnight dexamethasone suppression test: failure to suppress basal levels by 50% or a level >120 nmol/l suggests Cushing's syndrome.
- Urinary free cortisol (normal: 100–350 nmol/24 h).
- Low-level dexamethasone suppression test.

Tests for ACTH dependence
- ACTH assay (normal levels: <25 ng/l at 9 am and <10 ng/l at midnight).
- High-dose dexamethasone suppression test.
- Metyrapone – an enzyme involved in the final stage of cortisol sythesis.
- CRH stimulation of ACTH.
- Bilateral inferior petrosal sinus sampling for ACTH.

Imaging techniques

- Skull imaging to detect microadenomas: CT scan (50% detection), MRI scan (66% detection).
- Adrenals: CT, MRI, isotope scanning (NP59).

Ⓐ7

- Treatment aims to normalise cortisol levels and return the pituitary and adrenal gland function to physiological control.
- Trans-sphenoidal microadenectomy in Cushing's disease.
- Unilateral adrenalectomy in Cushing's syndrome for an adrenal tumour.
- Bilateral adrenalectomy is no longer the treatment of choice for Cushing's disease. This will lead to lifelong steroid dependence and one-third develop Nelson's syndrome due to an invasive pituitary tumour.

Ⓐ8 The surgical problems associated with Cushing's syndrome are: poor wound healing, susceptibility to infections, peptic ulcers, fractures, DVT and vascular accidents.

Your revision notes:

Adrenal mass

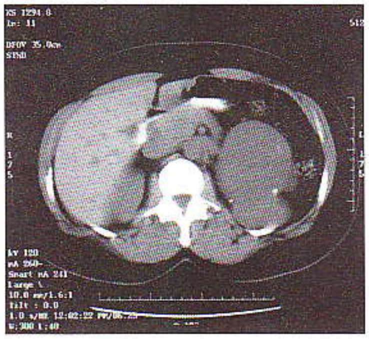


Figure 5.21

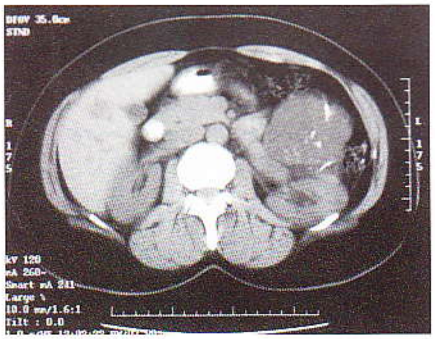


Figure 5.22

This 55-year-old patient had a routine CT scan to investigate long-standing abdominal pain. She has severe, treatment-resistant hypertension.

Questions

Q1 What is the abnormality on the CT scan?

Q2 What are the potential causes of this appearance on CT?

Q3 What is the differential diagnosis in this patient?

Q4 What further simple investigations might be appropriate?

Q5 Would you be more worried about malignancy if she was aged 65 years?

Q6 What are the treatment options for a patient in whom the lesion demonstrated secretion of aldosterone?

Q7 If the management involves surgery, what special precautions need to be taken pre-operatively?

Answers

(A1)

- Enlarged left adrenal gland.
- The adrenals are 'arrow-like' structures, located superior and anterior to the kidneys in the retroperitoneum.

(A2) This question essentially asks what are the causes of an enlarged adrenal gland. Causes can be divided up as usual:

Neoplastic

- *Benign*:
  - adenoma (may be silent or secreting, such as aldosteronoma or Cushing's tumour)
  - paraganglionoma
  - neurofibroma
  - phaeochromocytoma (although 10% are malignant, 10% extra-adrenal, 10% bilateral).
- *Malignant*:
  - adenocarcinoma – rare
  - metastasis (breast, bronchus, melanoma)
  - neuroblastoma (children only).

Cysts

- Usually small and asymptomatic, can cause diagnostic confusion, and they can become large.

Hyperplasia

- Unlikely as this is unilateral.

(A3) Essential hypertension is common so any of the above list may be possible, but in a hypertensive individual, the most likely differential diagnoses are the hormone-secreting tumours such as phaeochromocytoma, aldosteronoma (Conn's syndrome) and cortisol-secreting tumour (Cushing's syndrome).

(A4) Further investigations will be primarily to investigate the above syndromes:

- urinary catecholamines
- 9 am and midnight serum cortisol
- serum aldosterone and renin (recumbent and ambulant): aldosterone is high in Conn's syndrome, with failure of suppression on ambulation and undetectable plasma renin levels.

(A5) No – the risk of malignancy decreases with age.

(A6)

- A Conn's tumour should be resected if the diagnosis is made confidently (combination of radiology and biochemistry).
- Medical treatment of hypertension with spironolactone, an aldosterone antagonist (see answer to question 7).
- Open versus laparoscopic adrenalectomy:
  - open for large lesions or those with features of malignancy
  - laparoscopic is the standard approach and carries less risk of complications.

- For left adrenalectomy patient is placed lateral and ports placed:
  - transperitoneal approach
  - dissect the lienorenal ligament
  - trace the splenic vein laterally to find the adrenal vein and adrenal gland
  - ligate the veins and remove the adrenal.

Ⓐ7

- Hypertension and hypokalaemia present anaesthetic risk and both can be addressed by pre-operative therapy with spironolactone. Potassium supplements in refractory cases can be administered.
- Informed consent for laparoscopic adrenalectomy must include open conversion.

Your revision notes:

Phaeochromocytoma

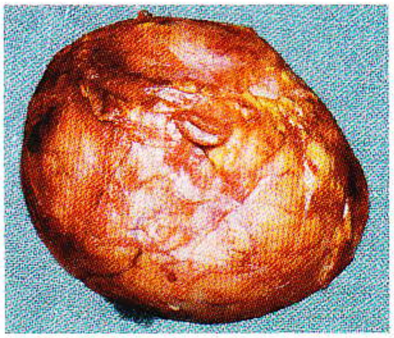


Figure 5.23

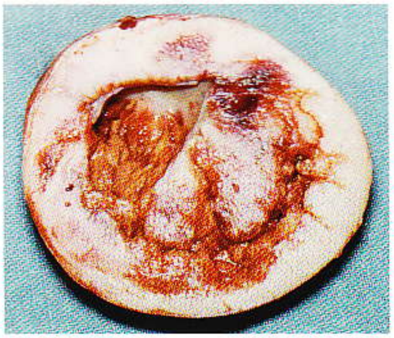


Figure 5.24

A 26-year-old male presented with episodic headache and a sensation of pounding in his chest. His BP was found to be 200/110 mmHg. This mass was subsequently removed from him after investigation.

Questions

(Q1) What is the possible cause for his hypertension?

(Q2) What are the sites of origin of these tumours?

(Q3) What are the clinical features?

(Q4) What are the associated conditions?

(Q5) How do you confirm the diagnosis?

(Q6) What are the important peri-operative measures in its management?

(Q7) What are the main principles involved during the surgical operation?

Answers

(A1) Phaeochromocytoma.

(A2) Phaeochromocytoma is a tumour of chromaffin cells which secrete catecholamines. Most (90%) arise from the adrenal medulla. The remaining 10% arise from extra-adrenal sites such as paraganglionic tissue of the sympathetic chain, and the organ of Zuckercandle. Approximately 10% are bilateral, extra-adrenal, multiple, familial and malignant. Paediatric tumours are more likely (60%) to be malignant.

(A3) Hypertension is the principal feature and this is paroxysmal in more than half of the patients. The paroxysms may be brought on by exercise, emotions, Valsalva manoeuvre, drugs and micturition. During the attacks the patient may have chest or abdominal pain and a pounding sensation. They are pale, sweaty and usually tachycardic. MI, pulmonary oedema, hypertensive encephalopathy, stroke, retroperitoneal bleeding, chronic cardiac failure or even sudden death may occur during a pressor crisis. It can also stimulate a diabetic or thyrotoxic state. It is a correctable cause of hypertension and accounts for 1–5 per 1000 new cases of hypertension.The biological actions of the secreted substances dictate the clinical signs. Adrenaline-secreting tumours (adrenal), which stimulate both alpha- and beta-receptors may cause tachycardia and hyperglycaemia. Noradrenaline-secreting tumours (extra-adrenal) activate peripheral alpha$_1$- and alpha$_2$-receptors leading to widespread cutaneous pallor and vasoconstriction. Pressor crisis may be associated with bradycardia.

(A4) MEN Type 2 (Sipple syndrome), von Recklinghausen's disease, von Hippel–Lindau disease, Sturge–Weber disease, tuberous sclerosis and acromegaly.

(A5) Diagnosis is confirmed by:

- *biochemical tests* to measure catecholamines and their metabolites, e.g. 24-hour urine collection for catecholamines and their metabolites such as VMA, metanephrines, homovanillic acid, 4-hydroxy-3-methoxy mandelic acid
  - normal plasma catecholamine levels: adrenaline, 0.1–0.3 nmol/l and noradrenaline, 0.5–3.0 nmol/l, measured by RIA
- *imaging*: adrenal CT and MRI – adenomas <10 mm can be missed. Venous sampling, MIBG scans (extra-adrenal tumours), 19-iodo-cholesterol (adenoma versus hyperplasia).

(A6) Careful control of hypertension is paramount.

- *Pre-operative medications*: initial alpha-blockers (phenoxybenzamine), beta-blockers (propranolol) only after alpha blockade, otherwise a hypertensive crisis can be precipitated. Metirosine is used to suppress catecholamine production.
- *Intra-operative medications*: sodium nitroprusside and phentolamine for hypertension and fluid replacement, dopamine and noradrenaline for hypotension. Handling of the tumour during its excision can result in profound hypertension followed by severe hypotension after its removal.

(A7) An open or laparoscopic technique. Both anterior and posterior approaches have been used in open surgery. The important surgical considerations are non-manipulative dissection of the tumour, early control of adrenal vessels, avoidance of capsular rupture, and clearance of adrenal fossa. If there is no appreciable fall in blood pressure after removal, every effort must be made to find multiple tumours.

Case 9

A patient with pain in the left flank radiating to the groin.

Questions

- Q1 What is the most likely diagnosis?
- Q2 What is the above test?
- Q3 What does it show?
- Q4 Would you expect this to resolve spontaneously?

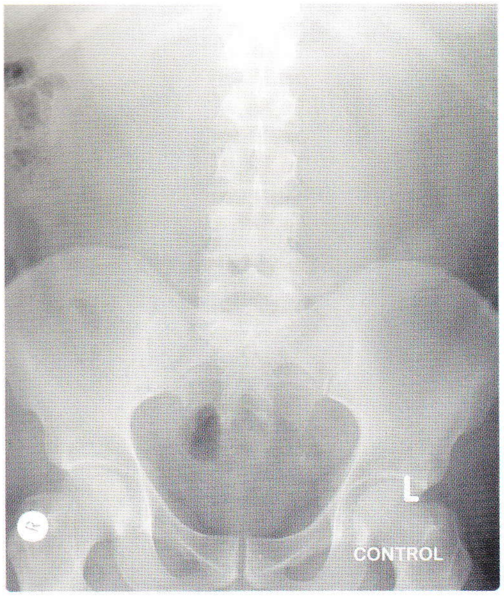


Figure 5.9a

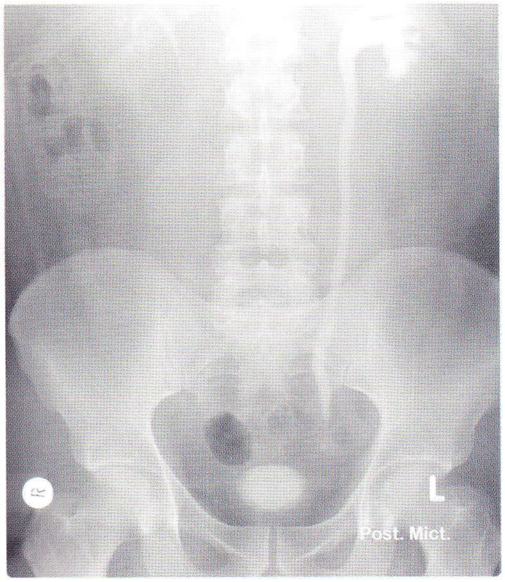


Figure 5.9b

Answers

(A1) Left-sided renal colic.

(A2) Intravenous urogram.

(A3) The control film shows a calculus within the left pelvis. The post-micturition film shows obstruction of the ureter with a standing column of contrast above the obstruction.

(A4) Yes – over 90% pass spontaneously.
Calculi < 4 mm pass in 90%.
Up to 8 mm in 50%.
Those over 8 mm rarely pass spontaneously and require treatment.

Case 10

A patient with left groin pain and night sweats, and raised inflammatory markers.

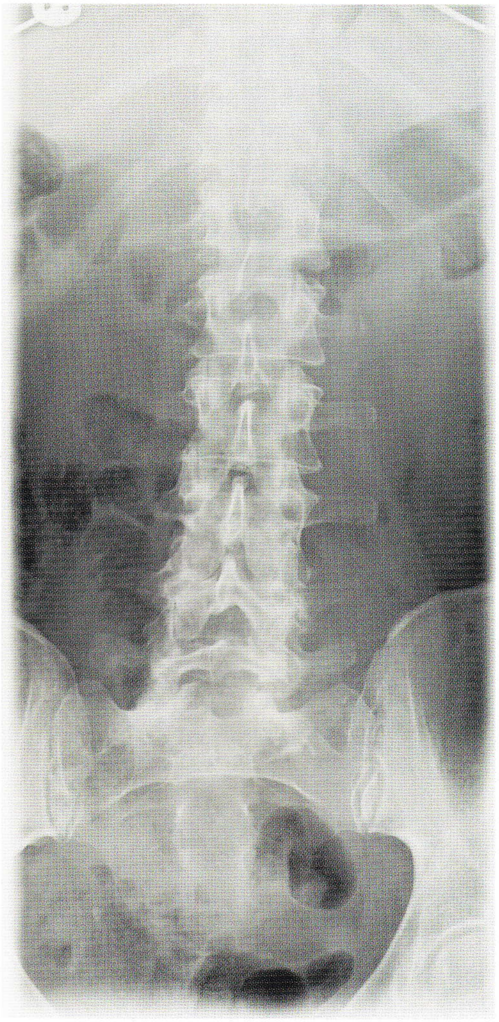


Figure 5.10a

Questions

Q1 What is the abnormality demonstrated in Figure 5.10a?

Q2 What is the most likely diagnosis?

Q3 What other type of imaging would be useful?

Answers

(A1) The right psoas shadow is bulging with a convex contour.

(A2) Right psoas abscess.

(A3) MRI.

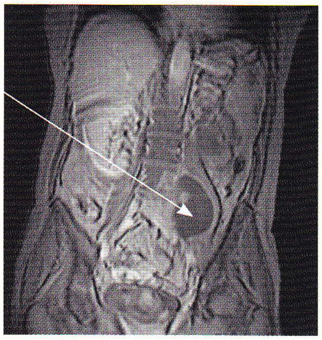


Figure 5.10b

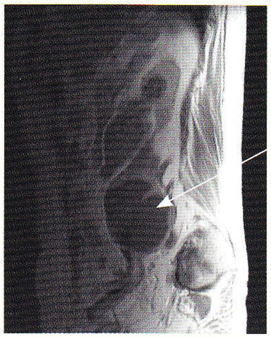


Figure 5.10c

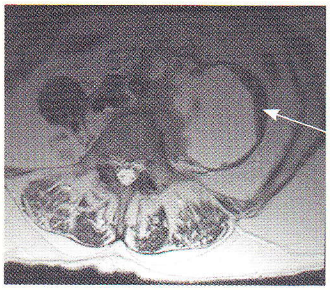


Figure 5.10d

Case 12

A patient with haematuria and recurrent urinary infection.

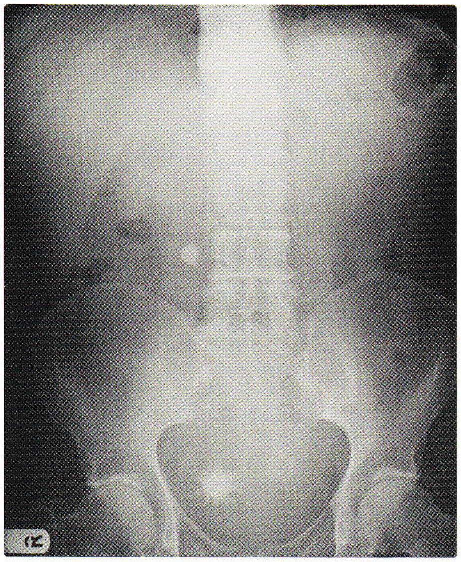


Figure 5.12a

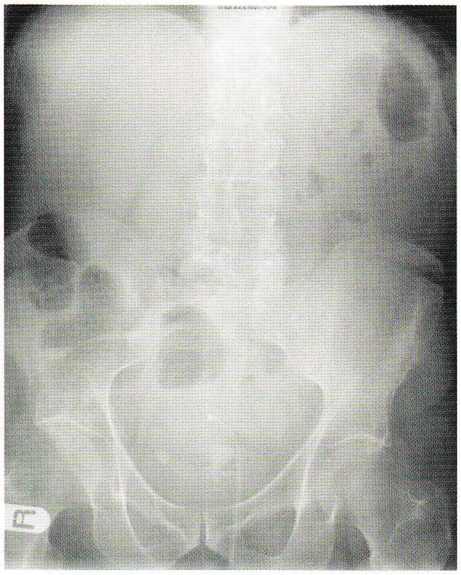


Figure 5.12b

Questions

Q1 What is the abnormality seen in both Figures 5.12a and b?

Q2 What are the different types?

Q3 If nothing is seen on the plain film does this rule out the diagnosis?

Q4 Are all types visible on radiology? If not, which ones are not?

Q5 Which are associated with infections?

Q6 Can they occur anywhere else in the urinary system?

Q7 How can they best be imaged?

Answers

(A1) Bladder stones seen within the pelvis.

(A2) Calcium stones.
Struvite stones.
Cystine stones.
Uric acid stones.
Xanthine stones.
Matrix stones.

(A3) NO – 30% are not seen. Some stones are radiolucent.

(A4) See above –
Struvite stones.
Uric acid stones.
Xanthine stones.
Matrix stones.

(A5) Struvite stones.
Matrix stones.

(A6) Yes – kidney and ureter.

(A7) Kidney – ultrasound.
Figure 5.12c shows a stone in the lower pole. It is echogenic with posterior acoustic shadowing.

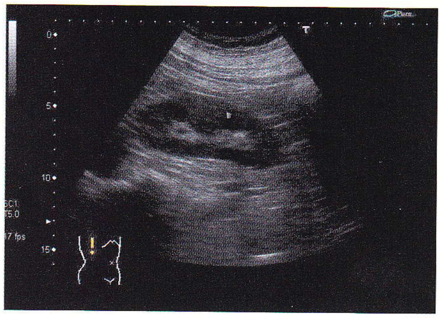


Figure 5.12c

Ureter – intravenous urogram – *see* Case 9.

Case 17

Figures 1.17a and 1.17b were taken during two different laparoscopic hernia repairs.

Questions

(Q1) What is the diagnosis in each case?

(Q2) What approach has been undertaken to repair these hernias?

(Q3) Identify the anatomy as indicated by the abbreviations.

(Q4) Where precisely has the mesh been placed in Figure 1.17c and what are the principles guiding this placement?

(Q5) Figure 1.17d demonstrates a number of right-sided herniae seen at initial laparoscopy. Identify each labelled defect.

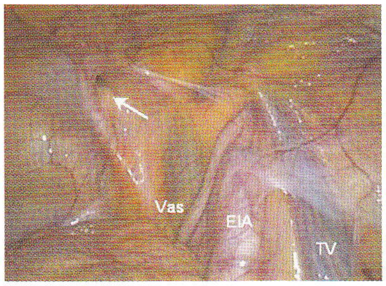


Figure 1.17a

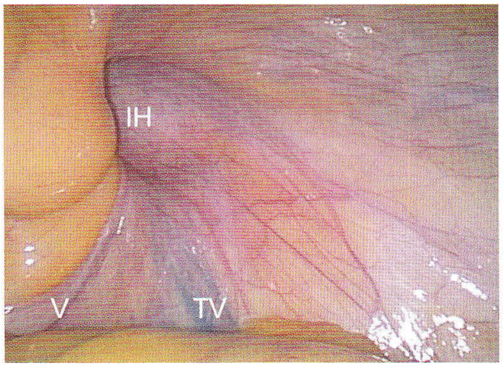


Figure 1.17b

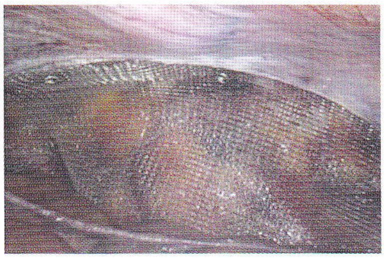


Figure 1.17c

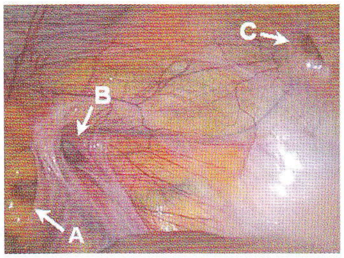


Figure 1.17d

Answers

Ⓐ1 Figure 1.17a: direct right inguinal hernia.
Figure 1.17b: indirect right inguinal hernia.

Ⓐ2 This is a TAPP repair (TransAbdominal PrePeritoneal).

Ⓐ3 Vas/V – vas deferens; EIA – external iliac artery; TV – testicular vessels.

Ⓐ4 The mesh is in the preperitoneal space. It should be big enough, usually 10 × 15 cm, to cover all potential weak spots (direct, indirect and femoral hernia sites) and reach the midline, as this is a common site of recurrence. Some surgeons fix it with tacks although there is evidence to show this may not be necessary. The peritoneum is closed over the mesh to prevent bowel adhesions.

Ⓐ5 A – Femoral; B – Indirect inguinal; C – Spigelian.

Section 3

Transplant and vascular access

Radiocephalic AV fistula

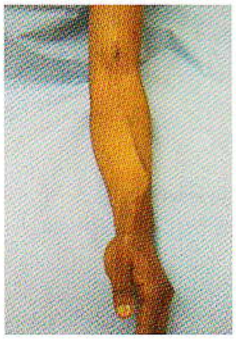


Figure 3.1

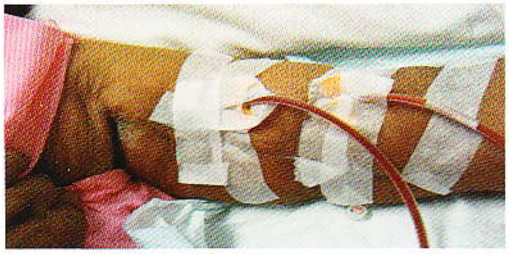


Figure 3.2

Questions

(Q1) What is the spot diagnosis and what is it used for? (Figure 3.1)

(Q2) Describe the clinical signs.

(Q3) What are the favoured sites for the formation of an AV fistula?

(Q4) Describe the AV fistula site shown in Figure 3.2 and other options available for access.

Answers

(A1) Radiocephalic AV fistula used as vascular access for haemodialysis.

(A2)
- *Inspection*: visible enlarged cephalic vein. If there is evidence of puncture marks from needling then this will confirm that this has been used for haemodialysis.
- *Palpation*: characteristic thrill or buzz 'like a mobile phone on vibrate mode'.
- *Percussion*: transmissible tap test.
- *Auscultation*: machinery murmur or continuous bruit.

(A3) Snuff box: radial artery to cephalic vein.
- The forearm: radial artery to cephalic vein, also known as a Brescia–Cimino fistula.
- The antecubital fossa: brachial artery to cephalic vein, also known as a brachio-cephalic fistula.
- The non-dominant hand is chosen preferentially, to free the dominant hand for use during haemodialysis sessions. The first fistula formed tends to be as distal as possible to preserve the vessels further up the arm for future access.

(A4)
- Brachiobasilic fistula between the brachial artery and the basilic vein. The upper arm basilic vein runs a deep course under the fascia above the antecubital fossa. This would therefore make it awkward to needle and position the needles for the haemodialysis sessions. It is therefore necessary to superficialise and transpose the basilic vein so that it lies in an accessible position in the forearm.
- Other possible sites/options include an autogenous vein and an artificial graft.
- An autogenous vein is preferred as there is a reduced risk of infection.
- If the cephalic vein is <2 mm then this is unlikely to mature to form a suitable fistula and it is appropriate to look at other veins. The basilic vein, which runs a superficial course in the medial side of the forearm, can be used:
  - the forearm ulnobasilic vein between the ulnar artery and the basilic vein
  - the forearm radiobasilic vein between the radial artery and the basilic vein. This will need mobilisation and transposition of the forearm basilic vein
  - the brachiobasilic vein as demonstrated can be used as an option.
- If the veins in the arms have been exhausted, then the next option is to look at the legs. The saphenous vein in the legs can be used either as an *in situ* graft from the popliteal artery, anastomosing the popliteal artery to the saphenous vein, or as a leg loop graft between the mobilised saphenous vein which has formed a loop to the femoral artery.
- If there are no suitable veins for access then the next option is to use an artificial graft such as PTFE or Dacron. There are some newer grafts which have been used for access. The sites can be anywhere that is superficial where an artery and a vein can be joined up with a graft.

Vascular catheters

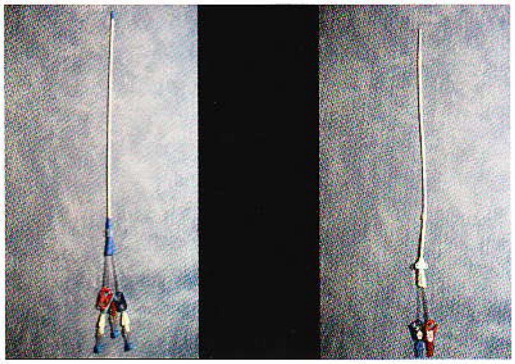


Figure 3.3

Questions

- Q1 What are these two instruments used for?
- Q2 What are the differences between the two catheters?
- Q3 Which vessels should be used for cannulation?
- Q4 What are the potential complications of insertion of these lines?
- Q5 What are the complications of an indwelling TDC?

Answers

A1 Vascular access catheters for haemodialysis.

A2
- One catheter has three lumens with no cuff and is used as a temporary means of providing haemodialysis (no more than 5–7 days).
- The second catheter is a tunnelled dialysis catheter (TDC) which has two ports and a cuff which serves two purposes. One is to secure the line by stimulating a fibrous response in the adjacent tissue of the subcutaneous tunnel, which then anchors itself to the catheter. The second purpose is to prevent migration of skin commensals and other bacteria to cause infection. For this reason the TDC can be used for dialysis for longer periods.

A3
- The right internal jugular vein is the vessel of choice since this has a direct route to the SVC and right atrium.
- If the left internal jugular is used then this will involve passing the cannula around the innominate vein, and the tip of both ports should be positioned in the SVC.
- The external jugulars can be used for insertion of a TDC, but final positioning of the tips needs to be in the SVC.
- The left and right femoral veins are then chosen.
- The subclavian veins should be avoided as there is an increased risk of stenosis, and this will compromise the ipsilateral arm for a more definitive access procedure.

A4
- Haemorrhage.
- Pneumothorax.
- Haemothorax.
- Air embolus.
- Cardiac arrhythmias.
- Inadvertent cannulation of the carotid artery.
- Traumatic AV fistula.
- Nerve injury.
- Malposition of the line.
- Malfunction of the line.

A5
- *Thrombosis*: each port needs to be primed with heparin at the end of dialysis to prevent this complication. If this fails then sometimes a thrombolytic agent such as tPA is used. It is essential to aspirate this before commencing dialysis, otherwise the patient will inadvertently be given a bolus which may render them coagulopathic.
- *Embolisation*: thrombi may form on the end of the catheter and propagate. This could cause a pulmonary embolus. If the ports are not primed or left open then they may develop an air embolus.
- *Mechanical*: if the TDC migrates or becomes kinked then it will no longer function. Sometimes the tip of one of the ports abuts against the side wall of the vessel and it simply needs to be pulled back.
- *Infection*: the catheter itself could become infected locally with tunnel site infections or infections of the catheter itself. This could rapidly progress to distant complications such as endocarditis, septic emboli and septicaemia. The management of this is to remove the catheter as soon as possible and treat with antibiotics. A new temporary line should be inserted into a different site after a few days of remaining free from prosthetic material.
- *Venous stenosis*: if left indwelling for prolonged periods there is also a risk of stenosis to the vein. This may progress to SVC occlusion but the main problem is the development of venous hypertension in the ipsilateral limb and compromised vascular access options in that limb.

PD catheters

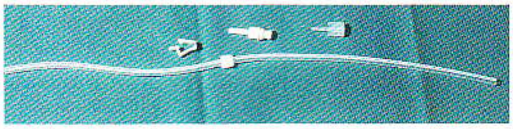


Figure 3.4

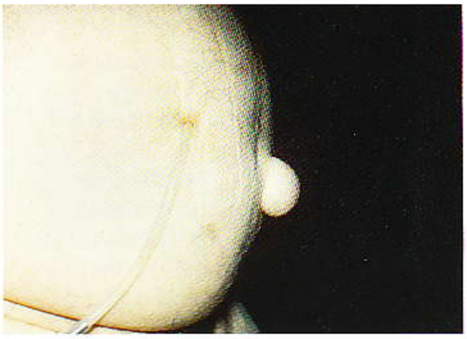


Figure 3.5

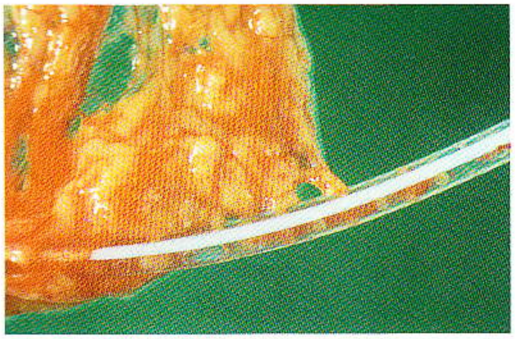


Figure 3.6

Questions

(Q1) What is the tube in Figure 3.4 called and what is it used for?

(Q2) Describe the features of the tube.

(Q3) How is renal replacement therapy carried out using this tube?

(Q4) What complications are demonstrated using this form of dialysis (Figures 3.5 and 3.6)? What other complications can occur?

(Q5) What are the relative benefits and disadvantages of this modality of dialysis compared to haemodialysis?

Answers

(A1) A Tenckhoff catheter, and it is used for peritoneal dialysis.

(A2) A hollow tube made from inert material (silastic) which at one end has multiple holes for draining fluid easily. There are two cuffs made of Dacron which form part of the tube to anchor the tube in position (at the pre-peritoneal site of entry into the peritoneum and a few centimetres from the skin exit site). These cuffs also prevent migration of skin commensals and other bacteria along the catheter.

(A3)
- The fluid and solute transport characteristics of the peritoneum, which serves as an endogenous dialysis membrane, are used.
- Access to the peritoneal cavity is achieved using this catheter, and the tip of the catheter is placed in the pelvis.
- A specified volume of dialysing fluid is instilled into the peritoneal cavity by gravity-induced flow. The fluid is then allowed to dwell for a period during which solute removal and ultrafiltration are achieved. The fluid is then drained and discarded. The process is then repeated.
- In continuous ambulatory peritoneal dialysis (CAPD) the patient exchanges the bags themselves and the dialysis continues 24 h per day with four to five exchanges per day. In continuous cycling peritoneal dialysis (CCPD) or automated peritoneal dialysis (APD), an automated cycling device is used to regulate and monitor the dialysate flow into and out of the abdomen, and four to ten exchanges are performed nightly over 8–10 h.

(A4) *Umbilical hernia formation* (Figure 3.5): the increased intra-abdominal pressure may predispose to the formation of herniae, subcutaneous leaks or even pleural effusions if there are congenital diaphragmatic herniae.
- Figure 3.6 demonstrates *occlusion of the tube by omentum*. This will result in failure of instillation of PD fluid or drainage of the fluid from the peritoneum.
- For this reason some centres practise omentectomy at the time of PD catheter insertion.
- *Other mechanical problems* can occur, resulting in failure of instillation or drainage of fluid. This could be due to kinking of the tube, external compression, for example by a constipated sigmoid colon and rectum, or the tip of the PD catheter may have flipped out from the pelvis making it more difficult to drain fluid.
- *Infection* of exit site, tunnel or peritoneum.
- *Malfunction* of dialysis: due to peritoneal membrane failure, lack of an adequate peritoneal membrane or due to the presence of numerous adhesions minimising the space in the peritoneum for fluid.

(A5)
- Maintenance of relatively constant blood or serum levels of urea, creatinine and electrolytes using PD.
- Better blood pressure control with PD.
- Dialysate is a form of nutrition.
- Promotes self-care and independence.
- Can allow a more liberal diet with PD compared to strict diet and treatment schedules.
- Haemodialysis allows a comparatively shorter treatment time.
- Haemodialysis allows ongoing social interaction for patients with little support.

Sclerosing peritonitis

This is a study of a 38-year-old male who has had three years of PD.

Questions

Q1 What is this investigation (Figure 3.7) and what abnormalities does it show?

Q2 Which clinical features might this cause?

Q3 The CT scans in Figures 3.8 and 3.9 were performed in the same patient. What are the radiological abnormalities?

Q4 What is the differential diagnosis?

Q5 What is the management?

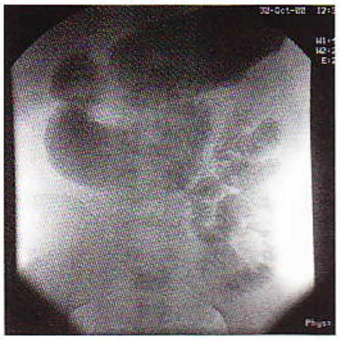


Figure 3.7

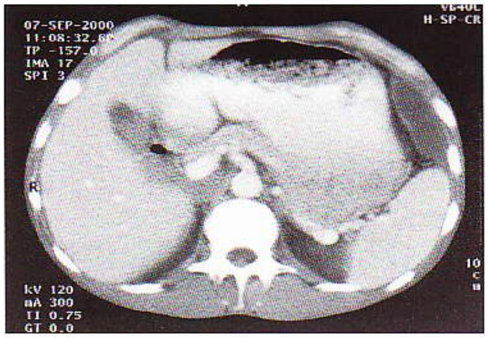


Figure 3.8

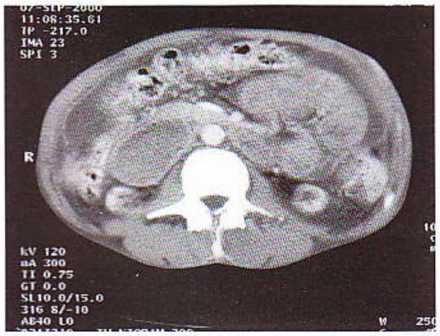


Figure 3.9

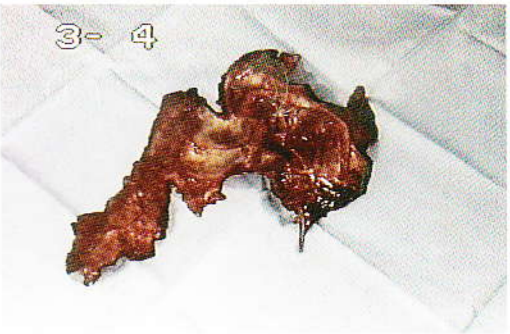


Figure 3.10

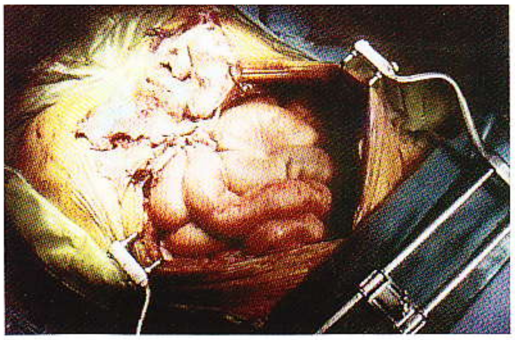


Figure 3.11

Answers

A1 This is a barium meal with follow-through which demonstrates a grossly distended stomach and duodenum.

A2
- *Symptoms*: vomiting undigested food products and bile, weight loss and failure to thrive, abdominal pain and distension.
- *Signs*: malnourished, dehydrated, tachycardia, abdominal distension, succussion splash.

A3 Cystic abnormality in the abdomen, thickened peritoneum and thickened small bowel.

A4
- Abscess.
- Peritoneal cyst.
- Mesenteric cyst.
- Pseudomyxoma peritonei.
- Peritoneal fluid collection which has become encysted.
- Sclerosing peritonitis.

A5 For sclerosing peritonitis the management is divided into conservative treatment, medical treatment with the use of azathioprine and tamoxifen, and surgical treatment. Laparotomy should be delayed until a period of hyperalimentation. The laparotomy findings often find a thickened peritoneum with chronic changes to the small bowel giving it a tanned, leathery appearance. There may also be encasement by a fibrous cocoon or rusk which can cause a more aggressive form of this condition known as sclerosing encapsulating peritonitis (see laparotomy findings in a different patient – Figures 3.10 and 3.11).
The cocoon is carefully removed from the serosa of the small bowel and any other fibrous tissue causing obstruction should also be excised.

Complications of AV fistulae

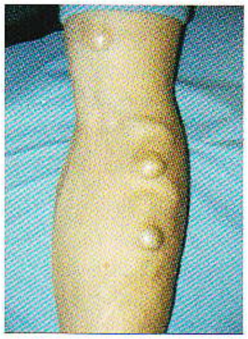


Figure 3.12

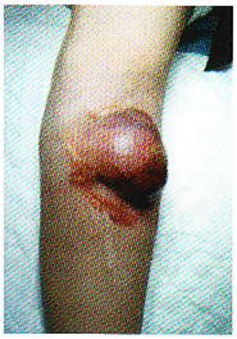


Figure 3.14

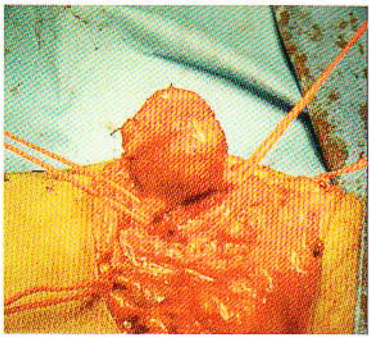


Figure 3.13

Questions

(Q1) What is the diagnosis for Figure 3.12 and what has caused this?

(Q2) What are the complications shown in Figures 3.13 and 3.14, and how can these be distinguished clinically?

(Q3) How should the conditions shown in Figures 3.13 and 3.14 be managed?

(Q4) What are the other specific complications of AV fistula formation?

Answers

(A1) Aneurysms of the arterialised vein. These have been caused by repetitive puncturing of the vein in the same site, known as 'buttonholing'. This technique should be avoided in artificial grafts as there would be a significant risk of rupture. Instead the site of needling should be staggered ('laddering').

(A2)
- In the aneurysm (Figure 3.13), the dilated part contains all three walls of the blood vessel (intima, media and adventitia), and the dilated portion has an expansile pulse.
- In the pseudoaneurysm (Figure 3.14), the wall of the dilated part contains clot and not the three layers of the blood vessel.

(A3)
- *Aneurysm* (Figure 3.13): if expanding significantly or causing other symptoms then the aneurysm should be resected. This could be performed either with ligation of the fistula or by attempting to preserve the fistula so that it could be used for subsequent access.
- *Pseudoaneurysm* (Figure 3.14): in the acute situation direct pressure over the leak with the main vessel may be of benefit. Sometimes an ultrasound probe may be used for direct pressure, with the added benefit of accurate visualisation of the abnormal communication. If this fails to control the aneurysm then radiological stenting may be of use or even open surgery to close the defect.

(A4)
- Failure to mature.
- Stenosis.
- Thrombosis.
- Venous hypertension.
- Steal phenomenon.
- High-output cardiac failure.
- Ischaemic monomelic neuropathy.

Renal transplant incision

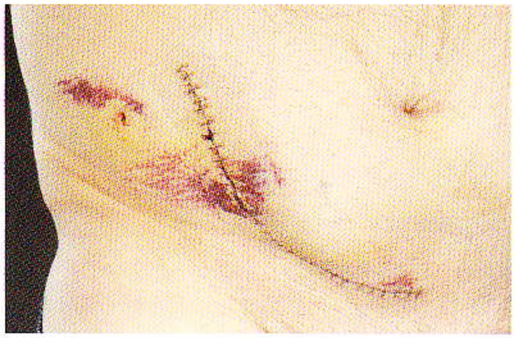


Figure 3.15

Questions

- (Q1) What is this incision and what is this used for?
- (Q2) What is the differential diagnosis of a mass in the right iliac fossa?
- (Q3) Is this a heterotopic transplant or an othotopic transplant, and what are the differences?
- (Q4) During this operation which anastomoses are performed?
- (Q5) From what donor source do these kidneys come?

Answers

(A1) A hockey stick incision for renal transplantation.

(A2)
- Kidney transplant – with accompanying incision.
- Ovarian tumour.
- Caecal tumour.
- Appendix mass.
- Iliac aneurysm.
- Inguinal hernia.
- Lipoma.
- Sebaceous cyst.
- Neurofibroma.
- Rhabdomyofibroma.

(A3) This is a heterotopic transplant as the new kidney is put into a different position to where the native kidneys are, and these are not removed.

(A4)
- Renal artery to external iliac artery or internal iliac artery.
- Renal vein to external iliac vein.
- Ureter to bladder.

(A5)
- *A living donor* who can be related or unrelated to the recipient.
- *A cadaveric or deceased donor*: these donors can be divided into brainstem death donors who will become heart-beating donors, and donors who do not fulfil the brainstem death criteria.

Renal transplant complicated by lymphocoele

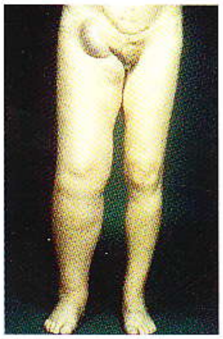


Figure 3.16

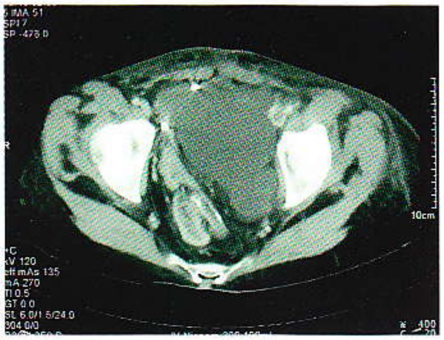


Figure 3.17

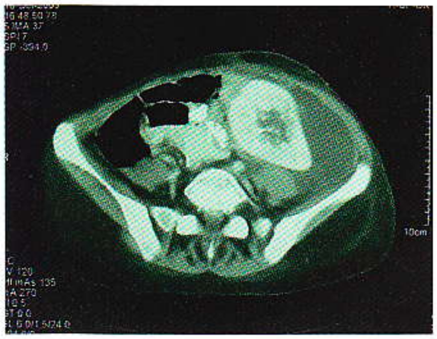


Figure 3.18

Questions

(Q1) What two clinical signs are seen in this patient (Figure 3.16) who has had a recent renal transplant?

(Q2) What is the differential diagnosis for these two clinical findings?

(Q3) How should a definitive diagnosis be made?

(Q4) Describe the radiological abnormalities (Figures 3.17 and 3.18) in this separate patient who had a transplant four weeks earlier.

Answers

(A1) There are two main clinical findings on inspection. These are the presence of a discoloured right groin swelling and the unilateral right leg swelling.

(A2) **Groin swelling**

- Inguinal hernia.
- Femoral hernia.
- Haematoma.
- Lymphocoele / seroma.
- Enlarged LN.
- Saphena varix.
- Abscess.
- Femoral artery aneurysm/pseudoaneurysm.
- Urinoma.

Unilateral leg swelling

- Deep venous thrombosis.
- Lymphoedema.
- Venous congestion caused by extrinsic pressure on the iliac femoral vein.
- Post-thrombotic leg swelling.

(A3) The diagnosis should be obvious after a thorough history, clinical examination and investigations of the groin swelling and leg swelling.

History

- Did the groin swelling precede the transplant?
- Did the leg swelling precede the transplant?
- Is there any previous history of deep venous thrombosis?
- If the groin swelling was present before the transplant, was it reducible? Did it have a cough impulse?
- Any other symptoms suggestive of pelvic compression such as urinary frequency or constipation?

Examination

- *Groin*:
  - inspection: cough impulse?
  - palpation: tender? warm? reducible? cough impulse? transmitted pulse? expansile pulse?
  - percussion
  - auscultation: bruit? bowel sounds?
- *Leg*:
  - inspection: chronic changes of venous hypertension?
  - palpation: warm? tender? pitting or non-pitting oedema?

Investigations

- An ultrasound of the groin swelling will be able to identify if the groin swelling contains fluid, and also the leg veins can be imaged to rule out a deep venous thrombosis. If a collection is present then this can be aspirated for biochemistry and microbiology. If there is doubt as to whether it is urine or lymph/serum, the creatinine level would give a fair indication of this. The urinary creatinine is >5 mmol/l whereas the level in lymphatic fluid is similar to that of serum (micromol/l). Furthermore, the glucose content in the serum/lymph would be the same as in serum, whereas it should be negative in urine (except in diabetics). Finally the protein content in lymph/serum should be >20 g/l compared to urine which should be <1 g/l.

- In this particular patient this was a cystic collection which contained lymph. The diagnosis was therefore a lymphocoele. There was venous compression in this patient but fortunately there was no deep venous thrombosis.

(A4) CT scan at two levels.

- At the level of the iliac crest there is a collection lateral to the transplant kidney. In the CT scan at the level of the femoral heads there are more abnormalities. There is a spherical circular collection which has caused compression and deviation of the bladder. Inside the bladder the urinary stent is seen. This central structure is also causing compression of the rectum. The iliac vein is also seen with evidence of thrombus within the lumen. In summary this collection has caused bladder and bowel compression and is likely to have contributed to venous stasis and caused a deep venous thrombosis. The presence of the ureteric stent has probably prevented ureteric obstruction of the transplant kidney.
- The collection in the image lateral to the kidney was demonstrated to be communicating with the deep pelvic collection (not shown) and the diagnosis was subsequently confirmed to be a lymphocoele.

Your revision notes:

Renal transplant complicated by renal vein thrombosis

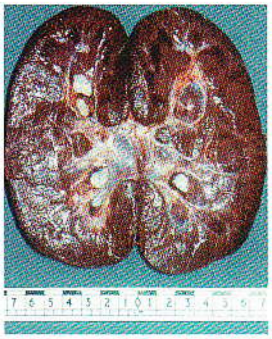


Figure 3.19

Questions

(Q1) What has occurred in this graft nephrectomy?

(Q2) What factors predispose to this?

(Q3) How may the condition present?

(Q4) What is the management?

(Q5) What is the differential diagnosis that could be prevented by a cross-match, and how is this performed?

Answers

A1 The specimen demonstrates a swollen congested graft nephrectomy with evidence of thrombus in the renal vein. This represents a renal vein thrombosis.

A2 This can be summarised by Virchow's triad:

1. *alteration in blood flow*: during a renal transplant there is stagnation of flow across the iliac vein when the vessels are clamped for the renal vein anastomosis. In the post-operative period, alterations in the blood pressure such as hypotension will cause reduced perfusion of the kidney
2. *alteration in the constituents of blood*: it is often observed that renal failure patients have a co-existing thrombophilia as evidenced by the coagulation of vascular access grafts. This could be due to a primary cause of hypercoagulability (such as protein C, protein S or antithrombin III deficiency or factor V Leiden mutation) or more commonly a secondary cause of hypercoagulability (such as antiphospholipid syndrome)
3. *damage to the vessel wall*: this is relevant during a renal transplant where the renal vessels could be damaged during the retrieval process, the benchwork preparation of the kidney or during the implantation. Care has to be taken to avoid trauma to the renal vein, with careful application of the vessel clamps to the iliac vein, minimal instrumentation of the intima during the anastomosis, and careful positioning of the venous sutures to avoid any stenoses or kinks that might be thrombogenic.

A3 The condition presents with a sudden decrease in the renal output in the early post-operative period. This may be accompanied with haematuria and pain over the renal graft. A duplex ultrasound would demonstrate renal vein thrombosis with reverse flow during diastole. If the condition progresses this could even lead to rupture of the kidney with a catastrophic loss of blood from the kidney.

A4 The condition occurs in approximately 1% of kidney transplants. The early diagnosis of a sudden loss of urine output with a duplex ultrasound may allow a surgical thrombectomy. Unfortunately at surgery there is little that can be done and this is managed with a graft nephrectomy. There is some evidence to suggest thrombolysis may have a role, but the risks of catastrophic bleeding from a recently formed anastomosis outweigh the potential benefits.

A5 Hyperacute rejection: this is a rare and largely preventable cause of immediate graft failure. It is caused by the presence of preformed antibodies in the recipient serum to donor antigens which would have developed by previous exposure to those antigens by blood transfusions, previous transplantation or pregnancy. This type of reaction can also occur if an ABO-incompatible transplant is inadvertently performed.

- There are two types of cross-match test:
  - *complement-dependent cytotoxicity* (CDC): lymphocytes (T and B) from the donor are incubated with serum from the recipient at room temperature, 37°C, and in the cold at 4°C. Rabbit complement is added to cause cell lysis. After 3 h of incubation the cells are inspected for evidence of killing. If the recipient's serum (in the presence of rabbit complement) kills the donor lymphocytes, then a preformed antibody is present and the cross-match is recorded as positive. This means the transplant is contraindicated
  - *flow cytometry*: donor lymphocytes are incubated first with recipient serum and then with a fluorescent-labelled polyvalent anti-IgG antibody. If the recipient antibody to donor lymphocytes is present, it will be detected by the flow cytometer. This cross-match test is more sensitive than the CDC test.

Renal artery stenosis

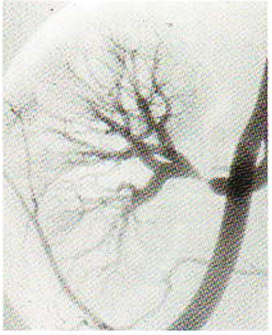


Figure 3.20

Questions

Q1 What is this investigation and how is it carried out?

Q2 What are the radiological findings?

Q3 How might this patient present?

Q4 Describe the non-invasive means of diagnosis.

Q5 How should this be managed?

Answers

(A1) Digital subtraction angiogram of a renal transplant: the ipsilateral or contralateral femoral artery is cannulated and a guidewire is inserted and manoeuvred into position. This is then exchanged for a further cannula, and radiopaque contrast is injected into the vessel.

(A2) This demonstrates that the renal artery is anastomosed to the internal iliac artery, but at the region of the anastomosis there is a stenosis.

(A3) The condition should be suspected in renal transplants where there was a difficult anastomosis with small, diseased or multiple donor vessels. The patient is likely to present with increasingly difficult to manage hypertension with the use of more antihypertensive agents. There may also be a deterioration in the renal function, with an elevated creatinine possibly precipitated by an ACE inhibitor. Occasionally there may be episodes of flash pulmonary oedema. On clinical examination there may be a new transplant bruit.

(A4) The clinical finding of uncontrolled hypertension on numerous antihypertensives with a new transplant bruit is highly suggestive of the diagnosis. Non-invasive investigations include a duplex ultrasound, MRA and a pre- and post-captopril renogram.

(A5) Transplant renal artery stenosis is diagnosed in approximately 2% of renal transplant recipients. The definitive diagnosis is made on intra-arterial DSA. Percutaneous transluminal angioplasty is the usual initial intervention and is successful in up to 85% of the procedures, but there is often a re-stenosis rate. If this fails then surgical reperfusion by re-implanting onto the internal iliac artery or a jump graft using the saphenous vein can be performed, but this is complex surgery due to the dense scar tissue around the transplant, and there is a risk of graft loss.

Laparoscopic organ donation

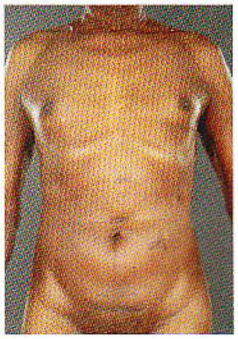


Figure 3.21

Questions

Q1 What procdure has been carried out in this patient?

Q2 What investigations should be carried out in this patient before deciding it is safe to proceed to surgery?

Q3 Which factors influence the side of surgery?

Q4 What are the benefits to the donor from this type of surgery?

Answers

(A1) Laparoscopic donor nephrectomy.

(A2) The donor has an extensive work-up prior to confirming suitability for donor nephrectomy and in practice this is divided into several phases:

- *phase 1*: initial assessment with blood group testing to ensure ABO compatibility, HLA tissue typing and cross-match to ensure immunological compatibility
- *phase 2*: history, examination and investigations of the potential donor including a comprehensive range of blood tests (haematology, biochemistry and virology) and urine tests (biochemistry and microbiology). The potential donor then undergoes a series of scans including an ultrasound (to confirm two kidneys), nuclear medicine GFR (to assess overall renal function), DMSA to check relative contribution to total function by each kidney
- *phase 3*: review of the above investigations and if OK an arteriogram is performed as a roadmap for the vasculature to both kidneys. This could be as an invasive DSA with selective cannulation of each renal artery to rule out anomalous vessels, or non-invasive, as a CT or MRI scan with three-dimensional reconstructions. A final cross-match between the donor and recipient is usually performed within a few weeks of surgery.

(A3)

- This can be influenced by donor factors, surgeon's preference and to a lesser extent recipient factors which would become apparent during the work-up process. However the basic principle is to accommodate the donor's wishes but also to prevent them from being potentially compromised in the future.
- The arteriogram determines the number of arteries to each kidney. Anomalous anatomy may make both donor nephrectomy and the recipient transplant more complicated.
- The DMSA scan may indicate a discrepant renal function between the kidneys, and if this is the case then the kidney with poorer function should be removed.
- If both kidneys have a single artery and vein with similar function, then the left side is preferentially removed on the grounds that it has a longer renal vein making the recipient procedure more straightforward.
- An ultrasound may demonstrate a benign abnormality in the kidney such as a simple cyst, which may influence the surgeon or the donor on the choice of side.
- Recipient factors that may influence the choice of graft nephrectomy in the donor could be the sex of the recipient (males have a narrow pelvis with deeper vessels), the weight of the recipient (if overweight then the surgeon will prefer longer vessels), and the proposed transplantation site (the iliac vessels tend to lie more deeply on the left side than the right).

(A4)

- There are numerous benefits to the donor which include reduced pain from the procedure, with fewer opiates required for a shorter duration, reduced hospital admission and earlier return to activities of daily living as well as work. The incidence of chest-related and wound-related complications is also thought to be reduced.
- There has been discussion of potential complications, but these have yet to be validated in a randomised controlled study. The potential complications are thought to be due to a prolonged ischaemic time between clamping the renal artery and flushing out the blood from the kidney with preservation fluid, which may lead to a delay in the kidney starting to work (delayed graft function). It has been postulated also that the laparoscopic process may potentially damage the renal vessels and the ureter.
- Although there is no doubt that there is a learning curve associated with the procedure, this should be regarded as a safe procedure in the hands of an experienced laparoscopic team.

Renal transplant with ureteric obstruction

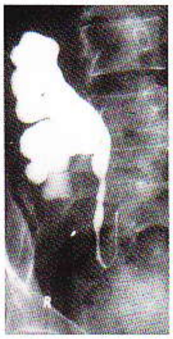


Figure 3.23

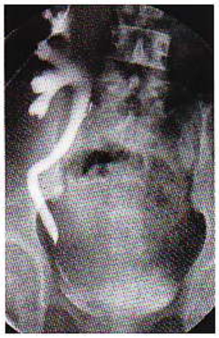


Figure 3.24

Questions

Q1 What is this investigation and what is the diagnosis?

Q2 Describe the radiological features of this investigation.

Q3 How is the diagnosis confirmed?

Q4 What are the causes of this abnormality?

Q5 How should the condition be managed?

Answers

(A1) This is an intravenous pyelogram and it demonstrates ureteric obstruction.

(A2) There is pelvicalyceal dilatation ('clubbing of the calyces') with dilatation of the ureter, which then comes to a stenotic segment probably at the vesico-ureteric junction. This is not a complete occlusion, as there is some contrast passing into the bladder.

(A3)
- The patient will present with impairment of renal function from the graft. The obstruction may be painless due to denervation of the graft.
- An ultrasound will demonstrate hydronephrosis although a mild degree of pelvicalyceal dilatation is often seen in renal transplant kidneys.
- An IVP as demonstrated may help confirm the obstruction as well as the site of obstruction, although good visualisation is dependent on reasonable graft function, and the contrast itself can cause a nephropathy.
- A retrograde ureterogram could be attempted but it is difficult to cannulate the transplant ureteral orifice.
- The most reliable means to confirm the diagnosis is by percutaneous antegrade pyelography. This also has the benefit of decompressing the obstruction and allowing recovery of the renal function, while planning a definitive procedure.

(A4) The causes can be divided into intraluminal, within the wall of the ureter, and outside the ureter.

Intraluminal
- Blood clots.
- Poor surgical implantation.
- Ureteral slough.
- Ureteral calculus.

Within the wall of the ureter
- Ureteral fibrosis secondary to ischaemia: the ureter receives arterial blood from the main renal artery and during the retrieval process this branch could be damaged, especially if the ureter has been skeletonised from its surrounding peri-ureteric fat.
- Ureteral fibrosis secondary to rejection: if the kidney is rejected, then the ureter may sustain an acute inflammatory injury resulting in fibrosis.
- Infection with polyoma BK virus is a rare cause of ureteric obstruction.

Outside the ureter
- The ureter could be compressed by kinking or twisting. It can also be compressed by haematoma, lymphocoeles and tumours.

(A5)
- Acute post-operative obstruction usually requires surgical repair. Most centres use a double J-stent to cover the procedure during the peri- and early post-operative period. These are usually removed at 8 weeks. It may be that any abnormality is observed only after this point.
- If the stricture is less than 2 cm long then it may be amenable to endourological techniques in an antegrade or retrograde approach. The stricture can be dilated with a balloon and then under direct vision the stricture can be incised with a cold knife. A stent can then be left in place for 2–6 weeks before being removed cystoscopically.

- If the stricture is more than 2 cm then this will be less amenable to percutaneous techniques and these strictures will require surgical repair (as well as those that fail endourological techniques). This will involve direct implantation of the ureter above the stricture into the bladder or if the stricture is too high then anastomosing the native ureter or bladder to the transplant renal pelvis. To facilitate the latter technique, a psoas hitch could be employed where the bladder wall is sutured to the ipsilateral psoas to reduce tension on the anastomosis. Alternatively a Boari flap could be performed where the bladder is opened with a rectangular incision (preserving one short side) to form a flap, which can be rolled to form a tube and anastomosed to the transplant renal pelvis.

Your revision notes:

Case 14

Victim of a road traffic collision.

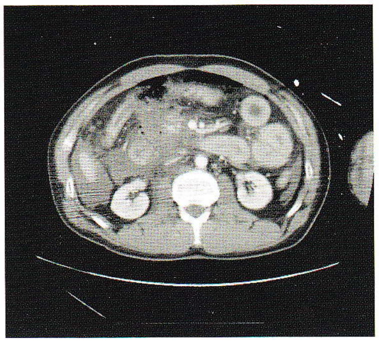


Figure 5.14a

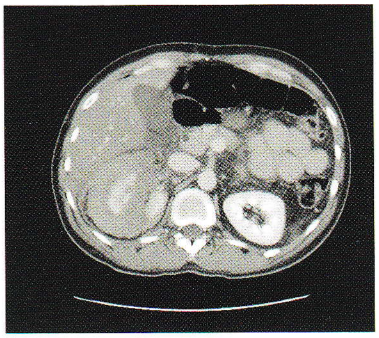


Figure 5.14b

Questions

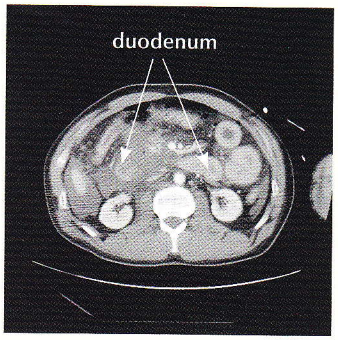
(Q1) What organs are involved in Figure 5.14a?

(Q2) What organ is involved in Figure 5.14b?

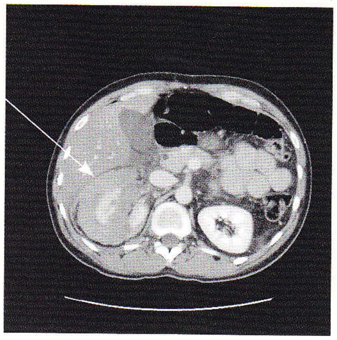
(Q3) What are the features in Figure 5.14a that indicate the patient is hypovolaemic?

Answers

(A1) The duodenum is ruptured.

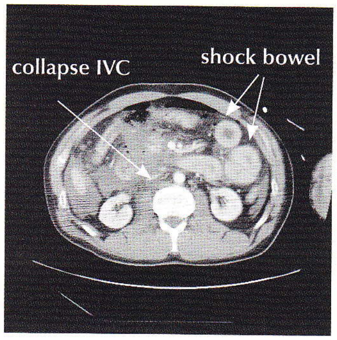


(A2) The right kidney is ruptured.



(A3) Collapsed IVC.
Shocked bowel:

- fluid filled, with thickened and enhanced bowel wall.



Abdominal compartment syndrome (ACS)

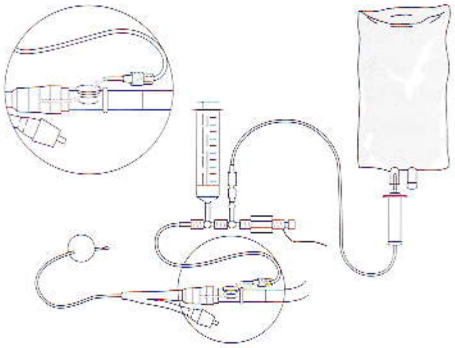


Figure 2.13

Questions

Q1 How is ACS diagnosed?

Q2 What is the cause of raised intra-abdominal pressure?

Q3 Under what circumstances should the diagnosis of ACS be entertained?

Q4 What are the possible means of diagnosing ACS?

Q5 What are the effects of ACS on various organ systems?

Q6 What levels of pressure cause concern?

Q7 What is the treatment? What is the risk of treatment?

Answers

A1 Intra-abdominal hypertension (IAH) is most often monitored with intravesical pressure measurement (as above) to diagnose ACS.

A2 The ACS represents the pathophysiological consequence of moderate to severe raised intra-abdominal pressure. The multiple trauma patient, with massive intra-abdominal or retroperitoneal haemorrhage and placement of intra-abdominal packs to control bleeding is a common cause. Severe intestinal oedema of whatever cause, or intestinal obstruction, and ascites under pressure are other well-known causes. Ruptured AAA patients, who have had prolonged surgery and large volume resuscitation, should also be kept in mind.

A3 When confronted with a patient with circulatory failure and elevated filling pressures – CVP or PCWP – it is imperative to exclude pericardial tamponade and increased pleural pressure (tension pneumothorax, status asthmaticus, etc). However the ACS should be kept in mind and excluded by pressure measurement.

A4
- A high index of suspicion should be kept in mind in the clinical scenarios mentioned above. However physical signs are misleading.
- Measurement of intravesical, inferior vena cava, rectal and gastric pressure reflects indirectly on IAP. Direct measurement of the IAP by direct puncture is sometimes possible but fraught with many problems and complications. In experimental conditions, bladder pressure is closely related to abdominal pressure and has become the most commonly used modality in clinical practice.

A5 **Cardiovascular system**

IAH has been shown to decrease cardiac output despite elevated CVP. Reduction in venous return to the heart appears a major problem. Volume challenge improves these parameters slightly, but only abdominal decompression results in significant improvement in cardiac function. Laparotomy with decompression markedly reduces CVP and PCWP and increases cardiac output.

Pulmonary and respiratory systems

Elevated peak inspiratory pressures, decreases in Pao_2 and increases in $Paco_2$ are often seen early on. The rises in peak airway pressure and intrathoracic pressure cause reduced venous return to the heart. The increase in airway pressures may also exacerbate pulmonary barotrauma. Peak airway pressure improves to near normal levels immediately after surgical decompression of the abdomen.

Renal system

Renal effects of IAH and ACS include decreased renal plasma flow and glomerular filtration rate, and oliguria or anuria. Improvement in renal function occurs only after abdominal decompression. It is suggested that the effects of IAH on renal function are related to compression of the renal parenchyma itself and to compression of renal vasculature, and are not only related to decreased cardiac output.

A6 IAP is normally 0 mmHg to subatmospheric, but may increase to 15 mmHg after abdominal surgery. IAH is defined as IAP higher than 15–20 mmHg (20–25 cmH_2O if a water manometer is used).

A7
- A laparotomy is done for decompression of established ACS. However patients are at risk of developing sudden, severe hypotension immediately after the abdominal cavity is opened. A sudden decrease in effective preload from re-expansion of abdominal and pelvic veins and from washout of anaerobic products is postulated.
- Anticipating the development of ACS and the use of an alternative wound closure technique, could prevent its occurrence.

Enteral nutrition

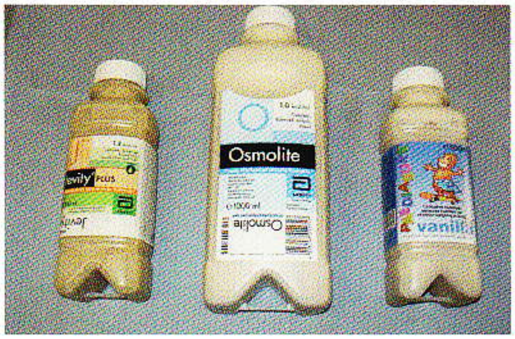


Figure 2.15

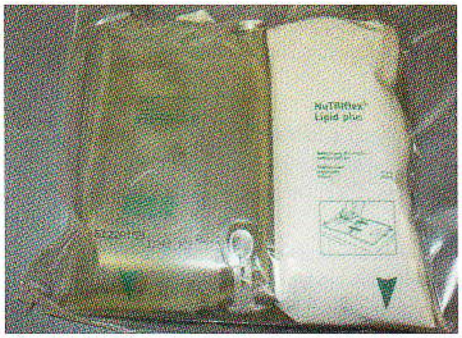


Figure 2.16

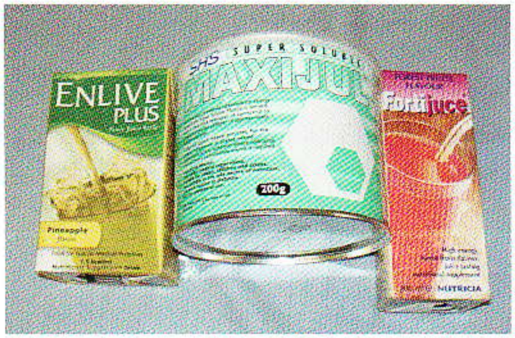


Figure 2.17

Figure 2.15 shows examples of enteral nutrition, Figure 2.16 examples of parenteral nutrition and Figure 2.17 examples of nutritional supplements. The following questions relate to enteral nutrition in the surgical patient.

Questions

(Q1) What are the advantages of enteral nutrition?

(Q2) What are the different ways to maintain enteral nutrition?

(Q3) What are some of the conditions where enteral feeding may not be possible (what are the indications for parenteral feeding)?

(Q4) What are the different types of diets that are given by the enteral route?

(Q5) What are some of the complications associated with enteral feeding?

Answers

A1
- Prevents mucosal atrophy.
- Reduces bacterial translocation from the gut, which has been considered as a cause for respiratory and other infections.
- Reduces the release of cytokines from unused gut.
- More physiological compared to parenteral nutrition.
- Avoids all complications of central line placement.

A2
- Normal oral intake.
- Fine-bore nasogastric feeding tube.
- Nasojejunal tube.
- Percutaneous endoscopic gastrostomy tube.
- Percutaneous endoscopic jejunostomy tube.
- Intra-operative placement of feeding gastrostomy/jejunostomy tube.

A3
- Intestinal obstruction.
- Non-availability of intestine as in short gut syndrome.
- In cases of enterocutaneous fistula.
- In the immediate post-operative period, in the presence of ileus.
- Where it is not possible to provide sufficient calories via the enteral route, the parenteral route may be additionally needed, as in severe trauma or in burns.

A4 Enteral feeds are broadly divided into three categories:
- *polymeric diets*: these contain whole protein as a nitrogen source, triglycerides and glucose polymers as an energy source, and standardised amounts of electrolytes, trace elements and vitamins. This type of diet is usually prescribed in patients with normal or near-normal GI function
- *predigested or elemental diet*: these contain free amino acids or oligopeptides as a nitrogen source, short-chain (>10) glucose polymers as an energy source and long- and medium-chain triglycerides as a fat source. These diets are preferred in patients with severe pancreatitis, or in short bowel syndrome
- *disease-specific diets*: these are specially formulated diets avoiding ingredients that can worsen an encephalopathy or respiratory failure.

A5
- Even though enteral feeding has many advantages, complications can result from feeding tube placement or directly related to the diet itself. Complications related to tube placement include malposition of the tube, unwanted removal and blockage of the tube.
- Diet-related complications include drug interactions, abdominal bloating, diarrhoea, cramps, regurgitation and pulmonary aspiration, and vitamin, mineral and trace element deficiencies.

Case I

The following pictures demonstrate the initial approach to a laparoscopic operation.

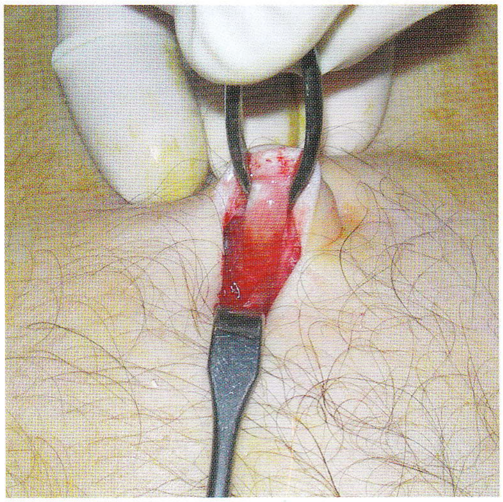


Figure 1.1a

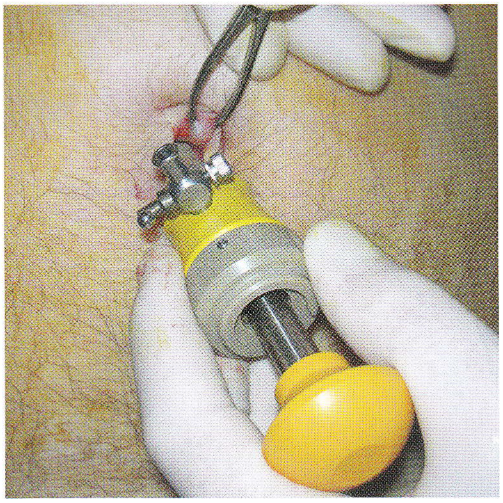


Figure 1.1c

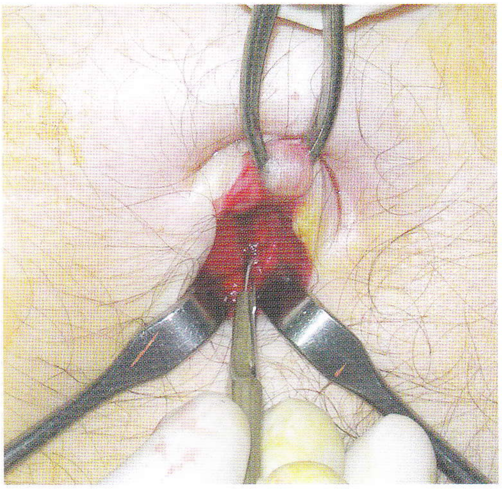


Figure 1.1b

Questions

Q1 Which technique for access to create a pneumoperitoneum is shown in Figures 1.1a–c?

Q2 Which anatomical structures need to be identified when applying it?

Q3 What alternative technique was widely used before this access?

Answers

(A1) The Hasson, or 'modified' Hasson technique.

(A2) The umbilicus and its insertion into the linea alba. The incision is made at this point, and the peritoneum punctured under direct vision to gain intraperitoneal access. A blunt trocar is then inserted and the abdomen insufflated with CO_2 under direct vision.

(A3) The Veress needle puncture. This is still used in some centres but concern has been raised over its technique of 'blind' puncture, which can result in increased risk of bowel or vascular trauma.

Case 2

Figure 1.2 shows part of a stack used in laparoscopic surgery, the 'endoflator'. It is important to know how this equipment works.

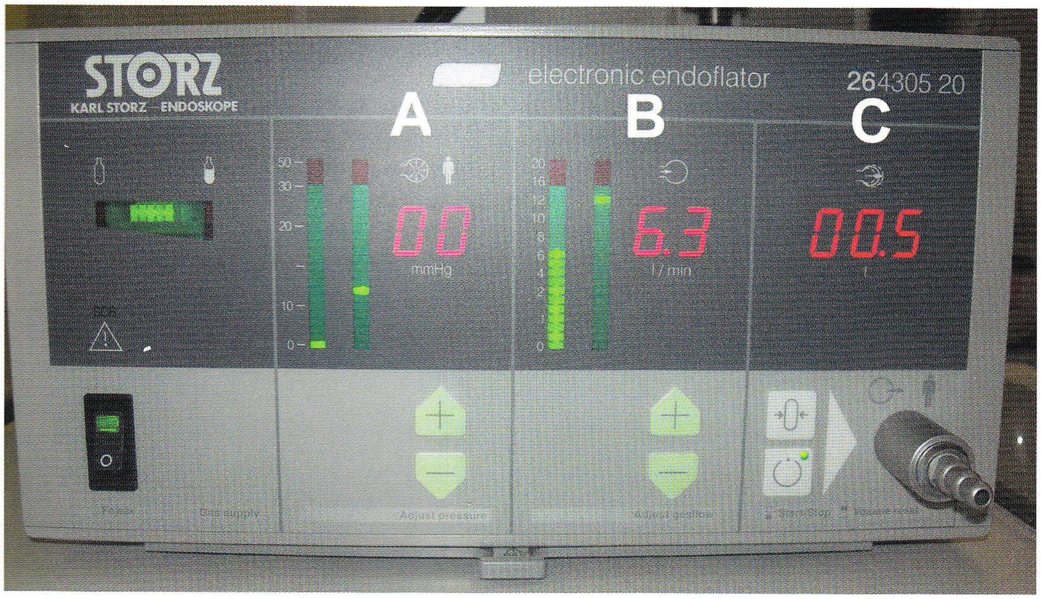


Figure 1.2

Questions

Q1 Describe the function of each of the columns labelled A, B, C.

Q2 What does the display to the left of 'A' indicate?

Q3 What might cause the left-hand LED in 'A' to rise sharply during surgery?

Answers

Ⓐ① This unit controls CO_2 insufflation. 'A' indicates the pressure level within the abdomen and allows the constant pressure to be set at a particular limit. Here it is set at 12 mmHg (right-hand LED). The actual abdominal pressure is indicated by the left-hand LED, zero at present as the unit is disconnected. 'B' indicates gas flow rate, usually set at 6–10 Litres/minute. Once the abdomen has reached its preset pressure the flow rate falls to zero. When gas escapes, reducing the pressure, this is detected by the unit and the flow restarts. 'C' indicates the total amount of gas used.

Ⓐ② This shows how much gas remains in the cylinder, and turns red when it is running low.

Ⓐ③ A sudden rise would be accompanied by an alarm signal, and indicates a sudden rise in the intra-abdominal pressure. A common cause of this is the patient beginning to wake up with the return of voluntary abdominal wall contraction (this is often an earlier 'warning signal' for the anaesthetist than the anaesthetic machine responses!). It may also happen with a mechanical block to the gas pipe (e.g. the tap is turned off).